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ROENTGENOGRAPHY IN THE LOCALIZATION OF BRAIN TUMOR, BASED UPON A SERIES OF ONE HUNDRED CONSECUTIVE CASES.

By George J. Heuer and Walter E. Dandy.

(From the Surgical Service of The Johns Hopkins Hospital.)

Roentgenography was first applied to the study of intracranial tumors in 1897 by Obici and Bollici [1] and, almost simultaneously, by Oppenheim.[2] They attempted to show that brain tumors cast shadows in the roentgenogram, using in their studies preserved specimens placed in the skull with or without the brain. They experienced little difficulty in obtaining shadows under these artificial conditions, but failed in their efforts to demonstrate tumor-shadows in the roentgenograms from living subjects. Nevertheless, Oppenheim,[3] although unsuccessful in his search for tumor-shadows, made an observation which indicated a more profitable field for study, namely, on the effects of brain tumor upon the skull; for, in 1889, he noted the destruction of the posterior clinoid processes of the sella turcica and made a correct diagnosis of an hypophyseal tumor from the roentgenogram.

Since the observations of Oppenheim a good deal of literature upon the X-ray diagnosis of brain tumor has appeared. For the most part it is widely scattered and consists of the citation of single cases showing tumor-shadows or deformations of the skull due to tumor; and, excepting the admirable monograph of Schüller,[3] and the special studies of sellar deformations (Fuchs,[4] Erdheim,[5] Giordani,[6] Jaugeas,[7] Köhler,[8] Fisher,[9] and subsequent writers), conveys but little idea of the value of the X-ray in the localization of brain tumor. We have, therefore, studied the roentgenograms in 100 patients presenting clinical evidences of tumor, with the purpose of discovering what abnormalities they show, and what value these abnormalities have in the focal diagnosis of brain tumor. We have found the presentation of the study difficult. In order to discuss variations from the normal, it is desirable to have in mind the X-ray picture of a normal skull. But, needless to say, perhaps no other bony structure presents so many apparently normal variations in shape and contour, not only of the skull as a whole, but of such individual structures as the sella turcica and the accessory sinuses, in the thickness and density of the bone and the prominence and distribution of its vascular supply, and in what are apparently pseudo-shadows, erosions or atrophic changes. The multiplicity of normal variations discourages the attempt to describe a normal skull as seen in the roentgenogram; and we shall merely indicate, in discussing the abnormalities due to tumor, the normal variations which may be confused with them. The difficulty of satisfactorily illustrating the text we have, in a measure, met by presenting with each untouched print a duplicate in which details lost in reproduction have been intensified. For this part of the work we are indebted to Mr. Brödel and Miss Norris; for the uniform excellence of the roentgenograms to Drs. Baetjer and Waters.

The present study, then, is based upon the first 100 patients with brain tumor who have come to the surgical service of Dr. Halsted since September, 1912. The statement, however, is not literally accurate, for we have included in the series patients with aneurism, pachymeningitis interna hemorrhagica,

gumma, tubercle, and arachnoiditis, who presented the clinical picture of tumor and whose roentgenograms showed abnormalities upon which we wish to comment. Ninety-six patients were operated upon, and through operation or subsequent autopsy we know the character and location of the lesion in 68.[10] We have a roentgenogram, and with few exceptions a stereoroentgenogram, of every patient. We have studied them rather from the viewpoint of the clinician than of the roentgenologist, using roentgenography as a laboratory aid in diagnosis in conjunction with a careful history and thorough neurological examination. Used in this way, abnormalities of the skull of significance to a roentgenologist must often be disregarded, because of historical and clinical data; and quite as frequently changes which of themselves are not impressive become significant when supported by physical signs. We may speak with some assurance of the roentgenological findings in the 68 patients in whom the location of the lesion is known; upon those of the remaining 32 patients we have hesitated to lay too much stress, although they have often been helpful in determining the diagnosis and operative procedures.

Regarded from the viewpoint of X-ray diagnosis, cerebral tumors may be roughly divided into three groups: (1) those which themselves cast a shadow in the X-ray plate; (2) those which, although casting no shadow, cause some deformation of the skull which we can recognize; and (3) those which give no evidence of their presence in the roentgenogram. The first group includes but few tumors, and in our experience only those that are bony or have undergone calcification, or, the uncalcified growths that have invaded the accessory sinuses. The majority of tumors belong to the second group, the roentgenograms showing not the tumor itself, but changes in the skull caused by its presence. These changes may be direct or local, caused by a tumor directly implicating a bony structure; or indirect, due to the pressure of the brain upon the interior of the skull caused by a tumor at a distance. We shall, then, discuss the X-ray manifestations of brain tumor under the following headings:

I. True tumor-shadows:
  (a) Uncalcified tumors.
  (b) Calcified tumors.
II. Changes in the skull due to tumor:
  (a) Changes in the skull due to general pressure:
    1. Enlargement of the skull.
    2. Separation of the cranial sutures.
    3. General convolutional atrophy.
    4. Destruction of the sella turcica.
  (b) Local changes in the skull:
    1. Local hypertrophy of the skull.
    2. Local expansion or enlargement without destruction.
    3. Local atrophic changes.
    4. Local convolutional atrophy.
    5. Local sellar destruction.
  (c) Vascular changes in the skull due to tumor.

This is obviously only a working classification, and a combination of two or more of the above manifestations may be present in a given case.

I. True Tumor-Shadows.

(a) uncalcified tumors.

It has been the experience of those interested in the X-ray diagnosis of cerebral tumors that uncalcified growths rarely if ever cast a shadow in the X-ray plate; apparently this is to be explained by the fact that their tissue density, even though considerable as in the endothelioma, is not sufficiently greater than that of the surrounding brain. The first tumor-shadow reported (Church, 1899 [11]), and two subsequently described (Mills and Pfahler [11]), were of uncalcified cerebellar and cerebral growths; and, if undoubted, are exceptional. With a possible reservation to be mentioned, we have no evidence that an uncalcified cerebral or cerebellar tumor casts a shadow while within the intracranial chamber. When, however, an uncalcified tumor invades the sphenoidal sinus, it may become visible in the roentgenogram; for the invading tumor-tissue is sufficiently denser than the air normally contained in this sinus to cast a shadow. In the roentgenograms of three patients we consider the shadows due to uncalcified tumor-tissue invading the sphenoidal sinus (Fig. 1). Two are cases of hypophyseal tumor in which the tumor has partially destroyed the floor of the sella turcica and entered the sphenoidal sinus. The third is one of tumor (sarcoma) arising in the nasopharynx, which by extension has invaded the sphenoidal sinus and become intracranial, producing general pressure symptoms. No intracranial shadow, however, is visible. We have no examples of extension of tumor-tissue into other accessory sinuses.

The reservation made above refers to seven certified cases of hypophyseal and two of suprasellar tumor, in the roentgenograms of which are definite shadows in or immediately above the sella turcica (Table I, Fig. 2). Since the tissue removed at operation has in four instances histologically shown calcification, we are inclined to consider the shadows in all to be due to areas of calcification. The point must be left unsettled for the present. The absence of similar shadows in the roentgenograms of our cases of certified temporal lobe tumor is against the possibility that they are due to uncalcified tissue.

Table I. Shadows Probably Due to Areas of Calcification in Tumors.

Assumption based upon four cases in which histological sections of tissue removed at operation show calcification in the tumor.

X-Ray No.	Location of lesion.	Verified at	Diagnosis.
15996	Hypophysis.	Operation.	Adenoma.
21639	Suprasellar.	Operation.	Glioma.
26735			
29912			
23254	Hypophysis.	Operation and autopsy.	Cyst.
26435			
29000	Suprasellar.	Autopsy.	Glioma.
30810	Hypophysis.	Operation.	Adenoma.
33036	Hypophysis.	Operation and autopsy.	Cyst; Rathke's pouch.
34943	Hypophysis.	Operation.	Adenoma.
35312	Hypophysis.	Operation and autopsy.	Malignant adenoma.
35359	Hypophysis.	Operation.	Adenoma.

Although these shadows in the sella turcica have little importance in the diagnosis of hypophyseal tumors, they may suggest glandular therapy in cases with a normal sella turcica and without neighborhood symptoms, but with glandular symptoms pointing to hypopituitarism, hypothyroidism, or their combination. A number of such cases, showing presumably extensive calcification of the hypophysis, have been observed, and a few have been definitely benefited by glandular therapy.

(B) CALCIFIED TUMORS (TABLE II, FIGS. 3-8).

True bony tumors (osteoma, osteosarcoma), or tumors which have undergone calcification or ossification, cast shadows in the X-ray plate that can be readily recognized; nevertheless, in a search of the literature we have found less than 20 recorded instances. We are aware that a number may have been overlooked, for, as we have observed, the literature is widely scattered. Lichtheim [12] was one of the first to demonstrate the shadow of a calcified tumor—a calcified gumma. The shadow was not recognized until the autopsy had disclosed the character of the tumor. Fittig [24] observed in the X-ray three calcified areas in the walls of cysts in an occipital-lobe glioma; Grunmach,[13] a shadow due to a calcified tumor of the pineal gland; and Algyogi,[33] a basal tumor with areas of calcification. Schüller [5] reported an osteoma in the frontal region and two cerebral tumors; Klineberger,[27] a tumor of the occipito-parietal region; Stieda,[28] a calcified cysticercus; and Sträter,[31] a calcified brain abscess, recognized after its disclosure at operation. Gottschalk [19] demonstrated a case of tumor-shadow before the German Roentgenological Society, but its reality was doubted by Holzknecht and Krause.[19] Presumably, many of these pseudo-shadows have been erroneously diagnosed as true tumor-shadows.

TABLE II. CALCIFIED OR BONY TUMORS CASTING SHADOWS IN THE X-RAY PLATE.

X-Ray No.	Location of lesion.	Verified	Diagnosis (Path.)
15517	Third ventricle. Extending into hemisphere.	Only by X-Ray.	Glioma (?).
30238	Hemisphere; Postcentral.	At operation...	Osteosarcoma.
30263			
16456	Temporal fossa, left..	At autopsy....	Aneurism, internal carotid arteries.
19594	Hypophysis.....	No.....	(?).
23529	Suprasellar. Third ventricle.	At operation and autopsy.	Cyst of Rathke's pouch. Calcification in cyst wall.
35387	Chiasm and optic nerves.	At operation...	Sarcoma.

The shadow cast by a calcified or bony growth varies with the nature of the tumor and the degree of bony or calcified change. It represents an area of increased density and is therefore of a lighter color than the surrounding negative. As commonly seen, the shadow of a solid tumor which has undergone extensive bony or calcified degeneration is quite dense but rarely of uniform density, owing to the scattered distribution of the bony or calcified areas. Its outlines may be quite definite; or indefinite and marked by islands of calcified tissue. Although of great value in determining the presence and location of a tumor, the shadow may give no idea of its size; for, since the process of calcification is progressive, a large tumor may be represented by a small shadow if the process be early, and may be completely outlined if the process be advanced.

Quite a different appearance may be presented by those shadows due to areas of calcification in the walls of cysts, abscesses and aneurisms. In place of a solid shadow they may appear in the roentgenogram as a series of incomplete ring-shadows that mark the boundaries of a more or less spherical mass. Most typically this type of shadow is seen in the wall of an aneurism that has undergone calcification (Fig. 5).

The localized areas of diminished density appearing as dark shadows in many roentgenograms are not to be confused with true tumor-shadows. They may be due to atrophy of the skull over a tumor and indicate the location of an uncalcified growth.

In six cases in this series, definite shadows are present in the roentgenograms. Three are quite large; two representing solid tumors, one an aneurism of the internal carotid artery. Two are small shadows representing bony or calcified areas lying upon the posterior clinoid processes; they indicate the location of the lesions, but, as subsequent operation or autopsy showed, give no information as to the size. The sixth is due to the calcification of an hypophyseal tumor. Through the kindness of Dr. Barker we have had the opportunity of seeing a seventh, not included in this series, due apparently to calcification in a cysticercus in the pre-Rolandic area; for cysticerci were removed from the subcutaneous tissues.

Owing to the infrequency of tumor-shadows these cases are presented in some detail.

1. Surgical No. 30803. Fig. 3. The roentgenogram shows a dense irregular shadow consisting of a central mass, 4 x 5 cm. in diameter, from which radiating shadows extend in every direction, but principally anteriorly and inferiorly. It lies 3 cm. below the vault, 4.5 cm. above the sella, and 6 cm. posterior to the frontal bone. When viewed stereoscopically, the tumor-shadow is seen to be in the right hemisphere near the midline. The lower radiating shadows approach the sella turcica, cross the midline, and extend to the region of the optic nerve of the opposite side. There is no visible destruction of the sella turcica.

Clinical Note.—A white male, 26 years of age, was admitted to the hospital November 14, 1912, complaining of loss of vision. His past history is uninteresting. His present illness began in November, 1909, with the accidental discovery of loss of vision in his right eye. An examination at this time showed, in addition, considerable impairment of vision in his left eye. Within a month he was compelled to give up his position because of failing vision. He later returned to work, although there was no improvement in his sight, and continued working for at least another year. He has had periodic headaches, not especially severe, and never associated with nausea or vomiting.

Examination. Positive Findings.—A large, well-nourished, healthy-looking man; normal intelligence and speech; total blindness in the right eye and a complete temporal hemianopsia in the left; primary optic atrophy of both discs; exaggerated reflexes

upon the left side of the body. There are no outspoken signs referable to the hypophysis.

Operative Treatment.—None. In view of the X-ray findings an attempt to remove the tumor appeared unwise, and the patient refused a palliative decompression for the relief of headaches.

Comments.—The insidious onset and rather inconspicuous general pressure symptoms indicate that the tumor has been of exceptionally slow growth. There is a long history of headache, dating from childhood, but no apparent change in its character or severity. From the roentgenogram the cause of blindness seems undoubtedly due to pressure of the calcified tumor upon the chiasm. The diagnosis of a supraseliar lesion could undoubtedly have been made from the history and the characteristic eye changes; the extent of the growth, however, could be determined only from the roentgenogram.

2. Surgical No. 37205. Figs. 4 and 4a. The shadow measures approximately 6 x 5 cm. in diameter, is dense, irregular in outline and presents minute isolated shadows at its periphery. Viewed stereoscopically, this calcified tumor seems to arise from the skull over the left parietal region and to extend well into the intracranial chamber.

Clinical Note.—A white, married female, aged 20, was admitted to the hospital June 9, 1915, with the complaint of headache and the presence of a tumor on her head. For five years she had noticed a tumor over the left parietal region, beginning as a small nodule and gradually growing until it measured roughly 7.5 x 6 cm. in diameter. A year ago she began to have headaches, general in character, but often frontal. These have increased in frequency and severity and on one or two occasions been associated with vomiting. There have been no other symptoms.

Examination. Positive Findings.—A bony prominence over the left temporo-parietal region measuring 7.5 x 6 cm. in diameter; double choked disc of 3 to 4 diopters; slight facial weakness on the right side; slightly increased reflexes upon the right side.

Operative Treatment.—A bone flap was outlined, the extent of which was guided largely by the roentgenogram. On lifting the bone the tumor was found tightly adherent to it and to the underlying dura. Therefore, before any attempt to elevate the bone flap, a dural flap was made. After elevation of the bone and dural flaps, the tumor, subsequently diagnosed as an osteosarcoma, was easily lifted out of a depression in the cortex. There were no adhesions between the surface of the tumor and the piaarachnoid of the hemisphere. The entire bone and dural flaps were removed. The dural defect was covered with a free transplant of fascia. The patient made a satisfactory recovery.

Comments.—A case in which the focal diagnosis could have been made perhaps from the prominence over the temporo-parietal region. The roentgenogram, however, showed us the character and size of the tumor and prevented us from making a mistake we had previously made of interpreting the protrusion as due to an osteoma of the skull.

3. Surgical No. 31628. Figs. 5 and 5a. Considered as a whole, the shadow in this case is of an entirely different character from the two preceding. It consists of a series of incomplete ring-shadows rather than a single solid shadow. There are seven distinct shadows in an area measuring 8 cm. anteroposteriorly and 5.7 cm. vertically, the series of shadows lying in the temporal fossa and encroaching upon the region of the sella turcica. The autopsy showed that the shadows in the roentgenogram represented areas of calcification in the wall of an aneurism of the internal carotid artery.

Clinical Note.—A white, married male, aged 28, was admitted to the hospital March 3, 1913. (He had two subsequent admissions, in October, 1913, and July, 1914.) Complaint, headache and failing vision. The present illness began over four years before this first admission with dimness of vision in his left eye. Three months later he began to have violent headaches associated with vomiting. They continued for six months; then gradually disappeared. He had at this time, exophthalmos with blindness of the left eye, and internal strabismus. Then for a period of four years he was free from headaches and able to perform his duties as a government telegraph operator. After this period of comparative health, vision became impaired in his right eye, and, fearing blindness, he came to the hospital.

Examination. Positive Findings.—Exophthalmos of the left eye, of the non-pulsating type; no bruit on auscultation over the head; complete blindness of the left eye; impaired vision, with a tendency toward hemianopsia for colors, in the right eye; optic atrophy in the left eye; well marked choked disc in the right; weakness of all the extraocular muscles of the left side; motor and sensory disturbances of the left trigeminal nerve.

Operative Treatment.—A correct diagnosis of aneurism was not made, and with the supposed growth well located by the X-ray plate, an attempt at radical extirpation was suggested. This the patient refused. A simple subtemporal decompression was therefore done upon the right side for the relief of pressure symptoms. The relief was complete and the choked disc in the right eye completely subsided. The patient returned to work and remained at work for a period of 18 months. He was then again seized with a sudden terrific headache, became unconscious, was brought to the hospital as soon as possible, but died an hour after his admission.

Autopsy.—A view of the base of the brain (Fig. 5a) shows a large aneurism occupying the left temporal fossa and extending beyond the median line to the right. It was, at the time of removal of the brain, entirely covered by the dura and fixed to the bone at the carotid canal. Upon the right side is a second smaller aneurism, 3.5 x 2 x 2 cm. in diameter, which has eroded the floor of the sella turcica. The hypophysis lies between the two masses, much compressed and flattened. The large mass on being separated from the brain shows clearly the internal carotid artery entering and leaving it at almost opposite poles. The vessels are of about normal size. The mass measures 24 cm. in circumference and 8 x 7 x 7 cm. in its various diameters. Its surface is smooth and roughly spherical, with the exception of three projecting knobs. On section, the wall of the aneurism varies from 1 cm. in thickness to paper thinness, and shows numerous areas of calcification. The sac is entirely filled with a laminated clot which can be lifted out *en masse;* the aneurism therefore being entirely solid except for a narrow channel extending through the clot. The base of the brain, after removal of the mass, shows an almost complete destruction of the tip of the left temporal lobe. The midbrain is compressed and dislocated to the right. The optic chiasm is rotated through an angle of 90 degrees, so that it lies flattened against the mesial aspect of the frontal lobe. On section of the brain the right ventricle is dilated; the left, collapsed. The tip of the left temporal lobe has undergone an extensive cystic degeneration.

Examination of the skull after removal of the brain shows complete destruction of the posterior wall of the left orbit, destruction of the left anterior and both posterior clinoid processes, partial destruction of the right anterior clinoid process, and erosion of the floor of the sella turcica.

Comments.—One of the most interesting features of this case is the X-ray picture. As previously noted, the calcified areas are in the form of more or less concentric, thin lamellæ. In retrospect, we should have suspected aneurism, for, in reviewing the cases in the literature and our own cases of calcified tumor, we have found no instance in which the calcification of a true tumor has been of this nature.

4. Surgical No. 34653. Fig. 6. The roentgenogram shows a small spherical area .8 cm. in diameter situated immediately above and anterior to the posterior clinoid processes. So close is it situated to these processes that it might readily be mistaken for their continuation. Stereoscopic examination, however, demonstrates its independence. The sella turcica is perhaps slightly enlarged. The case, verified by autopsy, is one of cyst arising from Rathke's pouch, the shadow in the roentgenogram representing an area of calcification in its wall. It illustrates the point that a shadow in the roentgenogram may give no idea of the size of the lesion.

Clinical Note.—A white, married female, aged 20, was admitted to the hospital May 25, 1914, complaining of blindness. The present illness began two years previously with severe nocturnal headaches. These were periodic, gradually increasing in intensity; occasionally accompanied by vomiting. Soon after the onset of the illness dimness of vision was noticed. This alternated with periods of normal vision until two months before admission. Since that time vision has rapidly declined, and on admission she was totally blind.

Examination. Positive Findings.—Blindness in both eyes; primary optic atrophy; the typical glandular picture of Fröhlich's dystrophia adiposogenitalis.

Operative Treatment.—A diagnosis of suprasellar lesion was made. By an intracranial hypophyseal approach a large cyst was exposed lying directly behind the optic chiasm. This cyst was evacuated. The patient recovered from the operation. Vision returned to a remarkable extent. The visual field in the right eye returned almost to normal; in the left eye there remained a temporal hemianopsia. The patient remained well for a year, and then returned with symptoms identical with those at her first admission. A second operation was performed. An attempt to remove the cyst wall was made. The patient succumbed.

Comments.—The X-ray in this case was of the greatest help in arriving at a diagnosis of a suprasellar lesion. The autopsy established the presence of a calcified area in the cyst wall quite independent of the posterior clinoid processes.

5. Surgical No. 39145. Fig. 7. The shadow in this case is almost identical with the preceding. It represents apparently a more or less spherical bony knob approximately .9 x .5 cm. in diameter lying upon the posterior clinoid processes. The sella turcica is more flattened than in the preceding case and the distance between the anterior and posterior clinoids greater. This case, verified at operation, is one of tumor of the optic nerve sheath.

Clinical Note.—A girl 21 years of age was admitted February 6, 1915, complaining of loss of vision. Her visual disturbance dates back seven or eight years, beginning with a blurring of objects, progressing to a hemianopsia, and finally to total blindness in the left eye; to a hemianopsia with marked impairment of vision in the right eye. She has had periodic headaches since the onset of her illness, never severe and not associated with nausea or vomiting.

Examination. Positive Findings.—A slender, normal-appearing girl; no acromegalic features; no signs of hypopituitarism; primary optic atrophy of both discs; complete blindness in the left eye; a temporal hemianopsia for form and colors in the right eye.

Operative Treatment.—An intracranial approach upon the left side exposed the chiasmal region satisfactorily. There presented between the optic nerves a hard, spherical tumor about 1.5 cm. in diameter, attached by a pedunculated base to the left optic nerve. After its attachment to the nerve had been cut across it was apparent that the tumor arose from the nerve sheath and that tumor-tissue extended into the left optic nerve. Further exposure showed that the right optic nerve and the chiasm were involved. The bony portion of the tumor shown in the X-ray was freely movable.

The patient is living. Her vision has remained stationary.

6. Surgical No. 33012. Fig. 8. The interest in the roentgenogram relates to the sella turcica. It is definitely outlined, measures 1.6 cm. in its antero-posterior diameter, and is about 1.4 cm. deep. The distance between anterior and posterior clinoids is increased. Within the sella turcica is a dense shadow, due apparently to the calcification of the entire hypophysis. Owing to this shadow it is difficult to make accurate statements regarding the anterior and posterior clinoid processes. The anterior clinoids are apparently intact; the posterior clinoids appear to have been destroyed. There is no destruction of the floor of the sella turcica. The sphenoidal sinus is unusually large, its greatest antero-posterior diameter being 4.5 cm., its greatest width 1.5 cm.

Clinical Note.—A white, married male, aged 44 years, entered the hospital October 9, 1914, complaining of loss of vision. The present illness began one year before admission with dimness of vision in the right eye and some disturbance of hearing in the right ear. He became totally blind in his right eye in March, 1914, and had marked impairment of vision in his left eye. In the past three or four months he has had attacks of vertigo and staggering; for three months has suffered from headache confined to the right occipital region; and for four months has had subjective numbness of the right side of the face.

Examination. Positive Findings.—A large, well-nourished man, totally blind in both eyes; a double choked disc of 4 diopters, with scattered hemorrhages upon the discs and retinæ; nystagmoid movements to the right; hyperesthesia to pain, touch, and temperature on the right side of the face; slight right facial weakness; complete deafness to air conduction in the right ear.

Operative Treatment.—The diagnosis was indefinite. Because of his total blindness we were unable to test his visual fields, and hesitated to make a diagnosis of a lesion about the chiasm. The clinical signs suggested a cerebellopontine lesion, and a cerebellar exploration was undertaken. The lesion was not found, either in the cerebellar hemisphere or in the cerebellopontine angles. The patient was discharged relieved of his headaches and with a subsidence of his choked discs.

Comments.—The lesion is not certified. In retrospect we are inclined to believe, in view of the positive shadow in the sella turcica, that the lesion is an hypophyseal one.

In this small number of cases (6 per cent) the X-ray establishes not only the diagnosis but also the location of the tumor. In the two cases of suprasellar lesion, the diagnosis, to one not familiar with the variations in the conformation of the posterior clinoid processes, would have been difficult perhaps from the X-ray alone. Indeed, one of these cases was sent to us with X-rays taken elsewhere which were reported to be normal. Yet in a careful examination of over 200 roentgenograms (tumor, epilepsy, etc.) we have in no instance seen a normal variation in the posterior clinoid processes which could be confused with the condition seen in these cases.

The roentgenogram which shows incomplete ring-shadows is of particular interest. The shadow as a whole differs so markedly from that in solid tumors that we should have been

able to make a differential diagnosis between a solid and a hollow lesion; and it is possible that in subsequent cases such a differential diagnosis may be made.

Shadows Due to Calcification of the Choroid Plexus, Pineal Gland and Meninges (Table III).— The discussion of roentgenographic shadows due to the calcification of brain tumors

TABLE III. SHADOWS DUE TO THE CALCIFICATION OF THE MENINGES, PINEAL GLAND AND CHOROID PLEXUS.

X-Ray No.	Structure casting shadow.	Lesion causing symptoms.	Location of lesion.	Verified at
23011	Choroid plexus.	Gumma of the dura mater.	Temporal region...	Operation.
23029				
34451	Falx cerebri...	Presumed tumor.	Unlocalizable	No.
34610				
21315	Pineal gland...	Glioma.....	Central, subcortical.	Operation.
20557	" "	Cyst.....	Paracentral.....	Operation.
18706	" "	Glioma.....	Temporal lobe.....	Operation.
21760	" "	Pachymeningitis interna hemorrhagica.	Hemisphere.....	Operation.
19549	" "	Glioma.....	Hemisphere.....	Operation.
24376	" "			
29058	" "	Endothelioma....	Paracentral.....	Operation.
29878	" "	Glioma.....	Optic thalamus....	Autopsy.
30039	" "			
35908	" "	Glioma.....	Parietal lobe, subcortical.	Autopsy.
15966	" "	Adenoma.....	Hypophysis.....	Operation.
21639	" "	Glioma.....	Suprasellar.....	Operation.
30810	" "	Adenoma.....	Hypophysis.....	Operation.
35312	" "	Adenoma.....	Hypophysis.....	Operation. Autopsy.
23090	" "	Arachnoiditis....	Over cerebellum...	Operation.
32428	" "	Aneurism.....	Basilar artery, pons.	Autopsy.
25217	" "	Presumed cerebral.	Unlocalizable.....	No.
35144	" "			
15280	" "	Tumor, metastatic.	Cerebellum.....	No.
17842	" "	Tumor.....	Presumed cerebellar.	No.

should not be dismissed without mention of shadows the result of calcification of structures normally present in the intracranial space; for, unless the character and usual position of such shadows are known, they might well be confused with tumor-shadows. In the roentgenograms of patients presenting clinical evidences of tumor we have observed shadows due to the calcification of the choroid plexus, pineal gland and falx cerebri.

Choroid Plexus (Fig. 9).—In the single case which we have had, the shadow is lobate, lying about 3 cm. above and slightly posterior to the petrous portion of the temporal bone. Seen in the single plate, the shadows of the two choroid plexuses are superimposed. With stereoscopic vision, two symmetrically placed shadows occupy corresponding positions in the two hemispheres. In an antero-posterior roentgenogram the shadows are projected to the upper inner angle of either orbit. The patient whose roentgenogram shows this shadow is living after the removal of a dural gumma. The shadow, therefore, has not been certified.* A similar shadow has been described by Schüller, who also interprets it as due to calcification of the choroid plexus.

Pineal Gland (Fig. 10).—The pineal gland seems especially prone to calcification. In the roentgenograms of 17 patients its shadow is round or oval, varying from small size to 4 or 5 mm. in diameter, and situated in the midline from 1.5 to 2.5 cm. directly above the temporal bone. We have identified this shadow with the pineal gland by autopsy studies. Although in our experience it has had no pathological or diagnostic significance, variations in its size or position might be of value in the diagnosis of tumors of or about the pineal gland.

Falx Cerebri (Figs. 11 and 11a).—A shadow due to an area of calcification in the falx cerebri is present in the roentgenogram of one patient. A single plate (lateral view) shows a definite shadow, crossed apparently on either side by the greatly dilated meningeal arteries, suggesting a calcified tumor in the precentral region. The stereoscope, however, and more certainly an antero-posterior roentgenogram, show it to be exactly in the midline; and, although not certified, it is in all probability due to an area of calcification in the falx cerebri.

Shadows due to the calcification of structures other than those mentioned undoubtedly occur. Attention has been called to calcification in the walls of the cerebral vessels, in Pacchionian granulations, in the meninges associated with epilepsy or following old hemorrhages. Our experience is too limited to warrant comment upon them.

Pseudo-shadows.—The term is applied to various light and dark areas in roentgenograms of the skull, which may be confused with true tumor-shadows. In our study we have often seen so-called pseudo-shadows. They occur most frequently in the temporal fossa and in the occipital, suboccipital and frontal regions. In our experience they have no significance. They can be readily distinguished from the shadow of a calcified tumor, and can scarcely be confused with circumscribed areas of diminished density, the result of atrophic changes in the skull over cerebral growths.

II. CHANGES IN THE SKULL DUE TO BRAIN TUMOR.

Our study early showed us that uncalcified brain tumors fail to cast shadows in the roentgenogram, and that calcified or bony tumors are rare (6 per cent). A study of the roentgenographic changes in the skulls of patients with brain tumor was therefore begun in the hope of deriving information of diagnostic value. Attempting to correlate the clinical and roentgenographic manifestations of brain tumor, we have grouped these changes into those due to *general pressure*—the result of a tumor anywhere within the intracranial space—and those due to *local pressure*—the result of a tumor within or in immediate proximity to the part affected.

(A) CHANGES IN THE SKULL DUE TO GENERAL PRESSURE. (FIGS. 12 AND 13.)

Those which can be recognized in the roentgenogram are: (1) Enlargement of the skull; (2) separation of the cranial sutures; (3) general convolutional atrophy; and (4) destruction of the sella turcica or atrophy of its posterior clinoid

* Since the preparation of this paper a second case of calcification of the choroid plexus has come under observation. The autopsy showed extensive calcification of the choroid plexus; and by post-mortem studies the shadow in the roentgenogram has been identified with this structure.

processes. The atrophy of the skull in the temporal region so often seen at operation cannot with certainty be recognized in the roentgenogram. The vascular changes in the skull, frequently a manifestation of general pressure, are considered under a special heading. Produced by the pressure of the brain against the inner table of the skull, these general pressure changes may be due either to cerebral or posterior fossa tumors. It should be remembered, however, that the mechanism of the production of increased intracranial tension in *cerebral* differs from that in *posterior fossa* tumors. In the former the increased pressure is commonly the result of an increase in the intracranial contents due to the growth of the lesion; in the latter it is caused, not so much by the lesion itself, as by the occlusion by it of the iter and the consequent production of an internal hydrocephalus. Clinically, it is recognized that increased intracranial pressure develops more rapidly and reaches a higher grade in posterior fossa than in cerebral tumors; a fact which is substantiated by our roentgenographic studies.

Undoubtedly age plays an important rôle in these general pressure signs. Obviously, separation of the sutures with consequent enlargement of the skull and atrophy of the inner table take place more readily in the young.

(1) *Enlargement of the Skull* (Table IV (1), Fig. 12).—The roentgenograms of nine patients show a definite enlargement of the skull. The oldest patient is 18 years of age. The condition is associated in every instance with separation of the cranial sutures and, in most, with thinning and general convolutional atrophy of the skull. These three phenomena of general pressure are therefore commonly associated. Of the nine patients, eight had cerebellar or cerebellopontine lesions associated with an internal hydrocephalus, and one had a suprasellar lesion with an obstructive hydrocephalus due to the occlusion of the third ventricle. Although, then, enlargement of the skull is most commonly seen in association with subtentorial lesions, it may occur in association with cerebral tumors causing an obstructive hydrocephalus. It may occur, though rarely, in cerebral lesions without an associated internal hydrocephalus, as two cases of cerebral abscess, not included in this series, have shown.

TABLE IV. GENERAL PRESSURE SIGNS OF INTRACRANIAL TUMORS AS SEEN IN THE X-RAY PLATE.

1. ENLARGEMENT OF THE SKULL.*

	X-Ray No.	Character of enlargement.	Location of lesion.	Verified at
1	29000	General enlargement.	Suprasellar (glioma)	Operation and autopsy.
2	15311 35824	" "	Cerebellum (cyst)	Operation.
3	15409	" " frontal+	Cerebellum (glioma)	Operation.
4	26471	" " "	Cerebellum (cystic gl.)	Operation.
5	34779 35472	" " "	Cerebellum (cyst)	Operation.
6	34287	" " "	Cerebellum (glioma)	Operation.
7	34713	" "	Cerebellopontine angle (endothelioma).	Operation.
8	33425	" " "	Pons (glioma)	Autopsy.
9	35931	" "	Cerebellum (glioma)	Operation.

* X-Ray Nos. 25954 and 35739, not included in this series, are examples of enlargement of the head, separation of the cranial sutures, and convolutional atrophy occurring in cases of certified cerebral abscess without an internal hydrocephalus.

(2) *Separation of the Cranial Sutures* (Table IV (2), Figs. 12 and 13).—Owing to the great variation in the prominence of the suture lines in apparently normal skulls, it has been difficult to differentiate between the slight grades of diastasis and normal variations. Attempting to be fair in our interpretation, separation of the sutures occurs in the roentgenograms of 57 patients. In those of 41 patients the diastasis is of such mild grade as to have little significance. Nineteen of these patients had cerebral tumors, 14 presumed cerebral tumors, 5 cerebellar, 2 presumed cerebellar tumors and 1 a tumor of the pons. In those of 16 patients the separation of

TABLE IV. GENERAL PRESSURE SIGNS OF INTRACRANIAL TUMORS AS SEEN IN THE X-RAY PLATE.—Continued.

2. SEPARATION OF THE CRANIAL SUTURES.*

	X-Ray No.	Sutures involved.	Degree.	Location of lesion.	Verified at
1	18706	F. P.	Slight	Temporal lobe (glioma)	Operation.
2	21760	O. P.	Marked	Hemisphere (pachym. int. hemorrhagica)	Operation.
3	19549 24376	F. P. and O. P.	Slight	Temporal lobe (glioma)	Operation.
4	25752	F. P. and O. P.	"	Temporal lobe (glioma)	Operation.
5	23610	F. P. and O. P.	"	Hemisphere (subcort. glioma)	Autopsy.
6	25610	F. P. and O. P.	"	Frontal lobe (endothelioma)	Operation.
7	25893	F. P.	"	Paracentral (glioma ?)	Operation.
8	23717	F. P.	"	Temporal lobe (cyst)	Operation.
9	29058	O. P.	"	Paracentral (endothelioma)	Operation.
10	27259	O. P.	"	Hemisphere (subcort. glioma)	Autopsy.
11	27982	F. P. and O. P.	"	Temporal (dural gumma)	Operation.
12	27975	F. P.	"	Hemisphere (subcort. glioma)	Operation.
13	29878 30039	F. P. and O. P.	"	Hemisphere (subcort. glioma)	Autopsy.
14	29329	O. P.	"	Frontal lobe (endothelioma)	Operation.
15	31938 31987	O. P.	"	Hemisphere (bilateral endothelioma)	Operation; autopsy.
16	35908	F. P. and O. P.	"	Parietal lobe (subcort. glioma)	Autopsy.
17	19594	F. P. and O. P.	"	Hypophysis	No.
18	23254 26435	F. P. and O. P.	Marked	Hypophyseal and suprasellar (cyst)	Operation; autopsy.
19	29000	F. P. and O. P. (all sutures involved).	"	Suprasellar (glioma)	Autopsy.
20	30810	O. P.	Slight	Hypophysis (adenoma)	Operation.
21	35312	O. P.	"	Hypophysis (adenoma)	Operation; autopsy.
22	35387	F. P. and O. P.	"	Suprasellar (sarcoma optic nerve)	Operation.
23	16386	F. P.	"	Cerebellum (glioma)	Autopsy.
24	15134	F. P. and O. P.	Marked	Cerebellum (cyst)	Operation.
25	15311 35824	F. P. and O. P.	"	Cerebellum (cyst)	Operation.
26	17276	F. P. and O. P.	"	Cerebellopontine (endothelioma)	Operation.
27	15409	F. P. and O. P.	"	Cerebellum (glioma)	Operation.
28	25057	F. P. and O. P.	Slight	Cerebellum (cyst)	Operation.
29	27041	F. P. and O. P.	"	Cerebellum (cyst)	Operation.
30	26471	F. P. and O. P.	Marked	Cerebellum (cystic glioma)	Operation.
31	34779 35472	F. P. and O. P.	"	Cerebellum (cyst)	Operation.
32	33072	F. P. and O. P.	Slight	Cerebellopontine (endothelioma)	Operation.
33	34287	F. P. and O. P.	Marked	Cerebellum (glioma)	Operation.
34	34713	F. P. and O. P.	Slight	Cerebellopontine (endothelioma)	Operation.
35	32428	F. P. and O. P.	"	Pons (aneurism, basilar)	Autopsy.
36	29896	F. P. and O. P.	Fairly	Pons (glioma)	Autopsy.
37	33425	F. P. and O. P.	marked	Pons (glioma)	Autopsy.
38	35931	F. P. and O. P.	Marked	Cerebellum (cyst)	Operation.
39	17519	F. P. and O. P.	Slight	Presumed cerebral	No.
40	17426	F. P. and O. P.	"	Presumed cerebral (metastatic)	No.
41	30112	F. P. and O. P.	Marked	Presumed cerebral	No.
42	20717	O. P.	Slight	" "	No.
43	21035	F. P.	"	" "	No.
44	15214	F. P. and O. P.	"	" "	No
45	20650	F. P. and O. P.	"	" "	No.
46	16590	F. P. and O. P.	"	" "	No.
47	28790	F. P. and O. P.	"	" "	No.
48	27017	F. P. and O. P.	"	" "	No.
49	28733	F. P. and O. P.	"	" "	No.
50	28872	F. P. and O. P.	"	" "	No.
51	30777	F. P. and O. P.	"	" "	No.
52	30365	F. P. and O. P.	"	" "	No.
53	35499	F. P. and O. P.	"	" "	No.
54	23907	F. P. and O. P.	Marked	Presumed cerebellar	No.
55	21728	F. P. and O. P.	"	" "	No.
56	15478	F. P. and O. P.	Slight	" "	No.
57	15635	F. P. and O. P.	"	" "	No.

* X-Ray Nos. 35739 and 25754, cases of certified cerebral abscess not included in this series, show marked separation of the sutures.

The fronto-parietal (coronal) and the occipito-parietal (lambdoid) sutures were particularly examined and our observations are confined largely to them.

the sutures is marked. Eight of these patients had cerebellar tumors, 2 presumed cerebellar tumors, 2 pontine tumors, 2 suprasellar tumors associated with internal hydrocephalus, 1 a cerebral and 1 a presumed cerebral tumor. It is evident from this analysis that well-marked separation of the sutures is an infrequent finding in roentgenograms of patients with cerebral tumor, but a common finding in patients with cerebellar or posterior fossa tumors.

(3) *General Convolutional Atrophy* (Table IV (3), Figs. 12 and 14).—The term is used in the sense of Spiller to describe a form of pressure atrophy of the inner table of the skull most characteristically seen in the roentgenograms of patients with "idiopathic" internal hydrocephalus. As seen

TABLE IV. GENERAL PRESSURE SIGNS OF INTRACRANIAL TUMORS AS SEEN IN THE X-RAY PLATE.—Continued.

3. GENERAL CONVOLUTIONAL ATROPHY.*

	X-Ray No.	Degree of atrophy.	Location of lesion.	Verified at
1	27259	Slight	Hemisphere (subcortical glioma).	Autopsy.
2	18261	"	Optic thalamus (glioma)	"
	19897			
3	16456	Slight, more marked frontal	Temporal fossa (aneurism)	"
4	27165	" " "	Hemisphere (arach. cyst)	"
5	21639	Well marked	Suprasellar (glioma)	Operation.
	26735			
	29912			
6	29000	Marked	Suprasellar (glioma)	Autopsy.
7	35312	Slight	Hypophysis (adenoma)	Operation; autopsy.
8	21779	Marked	Cerebellum (glioma)	Operation.
9	16386	Slight	" (glioma)	Autopsy.
10	15134	Marked	" (cyst)	Operation.
11	15311	"	" (cyst)	"
	35824			
12	15409	"	" (glioma)	"
13	23090	Slight	" (arachnoiditis)	"
14	27041	"	" (cyst)	"
15	34779	Marked	" (cyst)	"
	35472			
16	32428	Slight	Pons (aneurism, basil. art.)	Autopsy.
17	33425	Well marked	" (glioma)	"
18	35031	Marked	Cerebellum (cyst)	Operation.
19	31250	Slight	Cerebellopontine (endothelioma.	Operation; autopsy.
20	17426	"	Presumed cerebral (metastatic).	No.
21	21035	"	Presumed cerebral	"
22	15214	"	" "	"
23	21304	"	" "	"
24	23907	Marked	Presumed cerebellar	"
25	35499	Slight	" cerebral	"
26	21728	Marked	" cerebellar	"
27	15635	Slight	" "	"

* X-Ray Nos. 35739 and 25754, cases of certified cerebral abscess not included in this series, show slight convolutional atrophy.

in the roentgenograms of patients with brain tumor, it may be local or general. Since a cerebral tumor exerts its greatest pressure locally, at least in the early stages of its growth, it may cause a local convolutional atrophy of the skull—a condition which will subsequently be spoken of. General convolutional atrophy of the skull may be seen in the late stages of cerebral tumors or as the result of internal hydrocephalus due to posterior fossa lesions.

The roentgenograms of 27 patients show a general convolutional atrophy of the skull. In those of 16 patients it is slight or merely suggestive and, except as an indication of general pressure, has little value in diagnosis. Four of these patients had cerebral, 6 presumed cerebral, 5 cerebellar and 1 presumed cerebellar tumors. In those of 11 patients general convolutional atrophy is marked. Six of these patients had cerebellar, 2 presumed cerebellar, 1 pontine and 2 suprasellar tumors with an associated internal hydrocephalus. These findings again are suggestive, indicating that marked general convolutional atrophy is more commonly seen in subtentorial or in suprasellar tumors which have caused an internal hydrocephalus.

(4) *Destruction of the Sella Turcica or Atrophy of Its Posterior Clinoid Processes* (Table IV (4), Figs. 12 and 13).—A proper interpretation of roentgenograms of the skull is of particular importance in considering deformations of the sella

TABLE IV. GENERAL PRESSURE SIGNS OF INTRACRANIAL TUMORS AS SEEN IN THE X-RAY PLATE.—Continued.

4. DESTRUCTION OF THE SELLA TURCICA OR ATROPHY OF THE POSTERIOR CLINOID PROCESSES.

	X-Ray No.	Condition of sella.	Location of lesion.	Verified at
1	34779	Complete destruction	Cerebellum (cyst)	Operation.
	35472			
2	34287	" "	" (glioma)	"
3	35031	" " (practically)	" (glioma)	"
4	21760	Marked destruction	Hemisphere (pachym. int. hemorrhagica).	"
5	26557	Atrophy posterior clinoid processes	Paracentral (cystic glioma).	"
6	15451	" " " "	Hemisphere (subcort. glioma).	Autopsy.
7	25752	" " " "	Temporal lobe (glioma).	Operation; autopsy.
8	25610	" " " "	Frontal lobe (endothelioma)	Operation.
9	27259	" " " "	Hemisphere (subcort. glioma).	Autopsy.
10	16386	" " " "	Cerebellum (glioma).	Autopsy.
11	15311	Marked destruction	" (cyst)	Operation.
	35824			
12	31250	Atrophy posterior clinoid processes.	Cerebellopontine (endothelioma).	"
13	20112	Complete destruction	Presumed cerebral	No.
14	21304	Atrophy posterior clinoid processes	" "	"
15	23907	Marked destruction	" cerebellar	"
16	30777	Atrophy posterior clinoid processes	" cerebral	"
17	30365	" " " "	" "	"
18	15478	" " " "	" cerebellar	"
19	16456	Complete destruction	Temporal fossa (aneurism)	Autopsy.
20	18706	Atrophy posterior clinoid processes	" lobe (glioma)	Operation.
21	35255	Flattening post. clinoid processes	Presumed cerebral	No.

turcica; for we are rather prone to consider destruction of the sella turcica as indicative of hypophyseal or suprasellar lesions. Aside from these conditions, destruction of the sella turcica or atrophy of its posterior clinoid processes may take place as a result of the local pressure of cerebral tumors arising in the immediate neighborhood of the sella, or because of general increased intracranial pressure due to cerebral or subtentorial lesions. The roentgenograms of 21 patients, none of whom had hypophyseal or suprasellar tumors, show destruction of the sella turcica or atrophy of its posterior clinoid processes. In those of 8 patients, 4 of whom had cerebellar, 1 presumed cerebellar, 2 cerebral and 1 presumed cerebral tumors, there is a marked or complete destruction of the sella turcica. From the position of the lesions, the sellar destruction must be considered a general pressure phenomenon, in the patients with cerebellar tumor, possibly due to the distention of the third ventricle. In the patients with aneurism of the internal carotid artery the sellar destruction is probably due to local rather than general pressure. In the roentgenograms of 13 patients, slight changes in the sella turcica, *i. e.*, atrophy or disappearance of the posterior clinoid processes, is evident; 6 had cerebral, 4 presumed cerebral, 2 cerebellar and 1 presumed cerebellar tumors. It is perhaps noteworthy that these slight sellar changes are not associated with separation of the sutures

and general convolutional atrophy. In the patients with cerebellar, or with cerebral tumors lying at some distance from the sella turcica, atrophy of the posterior clinoid processes must be regarded as due to general pressure. In four patients the tumor lay in the subcortical tissues of the hemisphere immediately adjacent to the sella, and in them atrophy of the posterior clinoid processes may be considered a local pressure phenomenon.

To briefly summarize the changes in the skull due to general increased intracranial pressure, we may say that enlargement of the skull is not infrequently seen in the young and generally in association with an internal hydrocephalus due to a posterior fossa lesion. Separation of the sutures, general convolutional atrophy and destruction of the sella turcica are seen rarely and to but a slight degree in cerebral lesions; they are seen commonly and to a marked degree in cerebellar or posterior fossa lesions. Slight grades of these changes thus far in our own experience have had no focal diagnostic significance; marked grades, when occurring as they usually do in combination in the same case, indicate a subtentorial lesion which has caused an internal hydrocephalus. They may indicate a suprasellar lesion associated with an obstructive hydrocephalus.

It is especially in the differential diagnosis between cerebral and subtentorial tumor that the presence in the roentgenogram of general pressure signs is helpful. Yet they proved of no aid in the cases of two patients, blind on admission to the hospital, in whom the differential diagnosis lay between hypophyseal or suprasellar and cerebellar tumor. The histories suggested a cerebellar tumor; the clinical signs, total destruction of the sella turcica, optic atrophy and the syndrome of hypopituitarism, pointed to an hypophyseal or suprasellar growth. We explored the cerebellum of one patient in whom at autopsy we found a large suprasellar tumor; in the other, we found at operation a cerebellar cyst. Needless to say our inability to examine their visual fields was largely responsible for the difficulties in making a differential diagnosis.

(B) LOCAL CHANGES IN THE SKULL DUE TO BRAIN TUMOR.

The local effects of brain tumor upon the skull are commonly destructive, as seen in the local atrophy of the vault over a tumor or the destruction of the sella turcica; yet a local hypertrophy of the skull over a tumor may occur and, instead of destruction, the sella turcica may, at least in the early stages of disease, show an enlargement or hypertrophy as seen in acromegaly. The local changes in the skull which we have observed are: (1) Local hypertrophic changes in the skull; (2) local enlargement without destruction; (3) local atrophic changes in the skull; (4) local convolutional atrophy, and (5) local destruction of the sella turcica.

(1) *Local Hypertrophic Changes in the Skull* (Table V (1), Fig. 15).—The roentgenograms of five patients show a well-marked, circumscribed hypertrophy of the vault; in one responsible for a diagnosis of osteoma of the skull. In four patients operation showed that the hypertrophied bone lay

TABLE V. LOCAL CHANGES IN THE SKULL DUE TO INTRACRANIAL TUMORS.

1. LOCAL HYPERTROPHY OF THE SKULL OVER TUMORS.

	X-Ray No.	Condition.	Location and character of lesion.	Verified at
1	17554	Local hypertrophy over tumor.	Hemisphere cerebral tubercle...	Operation.
2	21035	Marked hypertrophy over frontal region: suggests Paget's desease.	Hemisphere presumed cerebral..	No.
3	25610	Local hypertrophy over tumor.	Hemisphere dural endothelioma.	Operation.
4	31938	" " " "	" " "	"
	31987			
5	35952	" " " "	" " "	"

directly over a superficial tumor; in one it failed to disclose the lesion. The condition apparently is rare, for in our study of the literature we have not seen it mentioned. The mechanism of its production is not clear. In three patients the lesion found at operation was a dural endothelioma; in one a superficial, solitary tubercle. In all, the dura lying between bone and tumor, while intact, showed a great increase in vascularity, as did the hypertrophied bone; and it seems possible that the local increase in the blood supply may explain the local hypertrophy of the skull.

(2) *Local Enlargement of Structures of the Skull* (Table V (2), Fig. 12).—Enlargement of the Internal Auditory Meatus: The herniation of the cerebellum into the foramen magnum and, occasionally, of the cortex into the foramina of exit of the cranial nerves, suggests that these structures may

TABLE V. LOCAL CHANGES IN THE SKULL DUE TO INTRACRANIAL TUMORS.—Continued.

2. LOCAL ENLARGEMENT OF STRUCTURES (WITHOUT DESTRUCTION).

	X-Ray No.	Structure involved.	Location of lesion.	Verified at
1	34943	Sella enlarged (acromegalic).....	Hypophysis (adenoma).....	Operation.
2	18706	" enlarged—wide.....	Temporal lobe (glioma).....	"
3	35387	" widened anterior and posterior clinoids separated.	Chiasm and optic nerves (sarcoma).	"
4	23529	Sella enlarged and deepened.....	Suprasellar (cyst Rathke's pouch).	Operation; autopsy.
5	35144	" enlarged	Presumed cerebral.....	No.
	25217			
6	15478	" "	Presumed cerebellar.....	"
7	18037	" "	" "	"
8	19594	" "	Hypophysis.....	"
1	29000	Internal auditory meatus enlarged.	Suprasellar (glioma).....	Autopsy.
2	30810	" " " "	Hypophysis (adenoma).....	Operation.
3	21779	" " " "	Cerebellum (cyst).....	"
4	15134	" " " "	" (cyst).....	"
5	15311	" " " "	" (cyst).....	"
	35824			
6	26471	" " " "	" (cystic glioma).....	"
7	34287	" " " "	" (glioma).....	"
8	32428	" " " "	Pons (aneurism, basil. art.).....	Autopsy.
9	35931	" " " "	Cerebellum (glioma)	Operation.
10	17519	" " " "	Presumed cerebral.....	No.
11	23907	" " " "	" cerebellar.....	"

undergo enlargement recognizable in the roentgenogram. Thus far, however, we have been unable certainly to recognize such changes; and our remarks will be confined to the internal auditory meatus. Henschen [20] was the first to observe a unilateral enlargement of the internal auditory meatus due to the local erosion of a cerebellopontine tumor. At the autopsy he noted that the internal auditory meatus on the affected side was larger than on the opposite one, and expressed the view, which he was later able to substantiate, that the enlargement should be recognizable in the roentgenogram. We have studied

the roentgenograms of our patients with the purpose of determining whether or not we could positively recognize the enlargement of the internal auditory meatus, and, if so, how often and under what conditions it occurs. It is definitely enlarged in the roentgenograms of 11 patients, 7 of whom had subtentorial lesions associated with X-ray signs of internal hydrocephalus, 1 a suprasellar lesion with signs of obstructive hydrocephalus, 1 an hypophyseal tumor and 2 presumed cerebral lesions. In the roentgenograms of patients with cerebellopontine tumor we have been unable to recognize a *unilateral* enlargement of the internal auditory meatus; and in our experience, therefore, its enlargement has not been of localizing value. It should be stated, however, that the roentgenograms were not taken with the idea of studying this region particularly.

Enlargement of the Sella Turcica (Fig. 16): In the roentgenograms of 8 patients there is a definite enlargement of this structure without atrophy of the clinoid processes and without alteration of the distance relations between them. Two patients had hypophyseal tumors and the sella turcica in both is of the type seen in acromegaly, 2 had suprasellar tumors, 1 a tumor of the temporal lobe near the sella and 3 presumed cerebral or cerebellar lesions.

(3) *Local Atrophic Changes in the Skull* (Table V (3), Fig. 17).—The local atrophy of the vault over an immediately underlying growth—a great diagnostic aid in the localization of brain tumor—has in our experience been of rare occurrence.

TABLE V. LOCAL CHANGES IN THE SKULL DUE TO INTRACRANIAL TUMORS.—Continued.

3. LOCAL ATROPHY OF THE VAULT OVER TUMORS.

	X-Ray No.	Condition.	Lesion.	Verified at
1	25610	Atrophy of vault at periphery of hypertrophic area.	Hemisphere (endothelioma).	Operation.
2	30238 30263	Atrophy of vault over tumor.....	Hemisphere (osteosarcoma).	"

It is seen in the roentgenograms of 2 patients among 15 who at operation had superficial dural or cortical tumors. Neither is satisfactory for illustration, and we have therefore taken a perfectly typical example from the files of the roentgenographic department of the hospital. The condition should not be confused with depressions often seen along the median line and marking the position of venous lakes. Atrophy or erosion of the orbital plate is demonstrable in the roentgenogram of the patient with aneurism of the internal carotid artery. Thinning of the vault over a superficial tumor, noted at operation upon several patients, and local erosion of the base of the skull, observed in two patients at autopsy, have not been demonstrable in the roentgenogram. Local atrophy of the suboccipital region in patients with cerebellar tumor has not been definitely recognized.

(4) *Local Convolutional Atrophy of the Skull* (Table V (4), Fig. 5).—This form of local atrophy of the vault we interpret as due to the local pressure of a subcortical tumor; the extent of the convolutional impressions upon the inner table being roughly proportional to the depth of the tumor below the surface. It is seen in the roentgenograms of 18 patients, and in all occurs most outspokenly in the frontal region. Thirteen patients had cerebral, 3 presumed cerebral and 2 cerebellar tumors. Operation or autopsy showed that in seven patients

TABLE V. LOCAL CHANGES IN THE SKULL DUE TO INTRACRANIAL TUMORS.—Continued.

4. LOCAL CONVOLUTIONAL ATROPHY OF THE VAULT.

	X-Ray No.	Region involved.	Location of lesion.	Verified at
1	19751	Frontal..	Hemisphere—subcortical—precentral.....	Operation.
2	15451	"	Optic thalamus (glioma).....	Autopsy.
3	21760	"	Hemisphere (pachym. int. hemorrhagica).....	Operation.
4	19549	"	Temporal lobe (glioma).....	"
5	24376			
*6	16456	"	Temporal fossa (aneurism).....	Autopsy.
7	23610	"	Hemisphere (subcort. glioma in lateral ventricle)..	"
8	27932	"	Temporal lobe (gumma).....	Operation.
9	29878 30089	"	Optic thalamus (glioma).....	Autopsy.
10	29329	"	Frontal lobe (endothelioma).....	Operation.
11	31938 31987	"	Paracentral region (endothelioma, bilateral)	Operation and autopsy.
12	35952	"	" " " "	Operation.
13	23254 26435	"	Hypophysis (cyst).....	"
14	30810	"	" (adenoma).....	"
15	17276	"	Cerebellopontine (endothelioma).....	"
16	20112	"	Presumed cerebral.....	No.
17	20650	"	" "	"
18	30365	"	" "	"
19	15280	"	" cerebellar.....	"

* Included also in Table IV (3).

the area of local convolutional atrophy was in the immediate neighborhood of the tumor; yet in the roentgenograms of only one or possibly two were we able to recognize by means of the stereoscope that the condition was *unilateral* and therefore of localizing value. We may say, therefore, that local convolutional atrophy is often the result of the local pressure of a cerebral growth in its early stages, that it may represent the beginning stage of a general convolutional atrophy, and that only rarely in our experience is it demonstrably unilateral.

(5) *Local Sellar Destruction* (Table V (5), Figs. 18 and 19).—This is characteristically seen in the roentgenograms of

TABLE V. LOCAL CHANGES IN THE SKULL DUE TO INTRACRANIAL TUMORS.—Continued.

5. LOCAL DESTRUCTION OF THE SELLA TURCICA.*

	X-Ray No.	Condition of sella.	Lesion.	Verified at
1	23254 26435	Enlargement and partial destruction....	Hypophysis (cyst).....	Operation; autopsy.
2	33036	Enlargement and partial destruction....	" (cyst).....	Operation; autopsy.
3	35312	" " complete destruction..	" (adenoma).	Operation; autopsy.
4	35359	" " " "	" (adenoma).	Operation.
5	30810	" " partial destruction....	" (adenoma).	"
6	21639 26735 29912	" " " "	Suprasellar (glioma)..	"
7	29000	Complete destruction.....	" (glioma)..	Autopsy.
8	15966	Enlargement and partial destruction....	Hypophysis (adenoma).	Operation.

* The roentgenograms of four patients with hypophyseal or suprasellar tumors included in Table V (2) show enlargement of the sella turcica but no destruction.

6 patients with hypophyseal and 2 with suprasellar tumor. In 7 the sella turcica is enlarged and partially or totally destroyed without the roentgenographic manifestations of general pressure; in 1 the presence of marked general pressure signs led us to explore the cerebellum before arriving at a

correct diagnosis. It is interesting to note that in the roentgenograms of four other patients with hypophyseal or suprasellar tumor there were slight changes of or about the sella turcica, but no sellar destruction—an indication of how mutually necessary to each other are clinical and roentgenographic signs in the diagnosis of these conditions. The importance of roentgenograms in determining the method of approach to these lesions will be discussed in a subsequent communication.

(c) VASCULAR CHANGES IN THE SKULL DUE TO BRAIN TUMOR. (Table VI, Fig. 20).

Vascular abnormalities in the skull, the result of brain tumor, manifest themselves in the roentgenogram by an enlargement of the diploetic sinuses or an excessive grooving of the bone, either of vessels normally present or of newly formed and abnormally distributed vessels. Obviously, increased intracranial pressure, regardless of the location of the lesion, may cause vascular stasis and a general enlargement of the cranial vessels demonstrable in the roentgenogram; a superficial tumor, a local increase in the number and size of the blood vessels of the adjacent dura and bone. In the roentgenograms of 50 patients we have noted an enlargement of the cerebral vessels, either a dilatation of the middle meningeal arteries or of the diploetic or extracranial sinuses of the skull. In those of 46 patients the dilatation is general and often of slight grade—an indication of increased intracranial pressure, but of no value in the focal diagnosis of brain tumor. It is seen most commonly in patients with cerebral or presumed cerebral lesions; more rarely in those with posterior fossa tumors. In the roentgenograms of four patients there is, as demonstrated with the stereoscope, a marked, in one, an enormous, *unilateral* dilatation of the cranial vessels—a finding of great value in the focal diagnosis of tumor. The roentgenograms clearly show a circumscribed network of vessels which drain into a single large vascular trunk. In all four patients the lesion at operation proved to be a dural endothelioma lying within the area outlined by the vascular network. In one, without other localizing signs, the unilateral vascular dilatation enabled us to make a positive focal diagnosis.

A stellate arrangement of the diploetic sinuses in the parietal region of the skull has been observed in the roentgenograms of eight patients. It occurs apparently regardless of the location of the lesion, and in our experience is merely a sign of venous stasis.

TABLE VI. VASCULAR CHANGES IN THE SKULL DUE TO INTRACRANIAL TUMORS.

	X-Ray No.	Vascular changes.	Location of lesion.	Verified at
1	17554	Enlargement of meningeal A, bilateral	Precentral (tubercle).....	Operation.
2	19751	Enlargement of diploetic vein, frontal.	Paracentral (subcort. gl.).	"
3	18706	Stellate diploetic veins, parietal region	Temporal lobe (glioma)...	"
4	23026	Marked gen. dilatation esp. frontal....	Frontal lobe (glioma).....	"
5	19549	" " " " "	Hemisphere (subcort. gl.).	"
	24376			
6	16456	Enlargement meningeal A, bilateral...	Temporal fossa (aneurism)	Autopsy.
7	25752	" " " "	Temporal lobe (glioma)...	Operation; autopsy.
8	25610	Enlarged vein from region of hyp. bone, unilateral.	Frontal lobe (endothelioma).	Operation.
9	23717	Enlargement meningeals and occipital vessels.	Temporal lobe (cyst arach.)	"
10	26856	Enormous dilatation (unilateral) of meningeal.	Occipital lobe (endothelioma.)	"
11	29058	General vascular enlargem'nt, bilateral	Paracentral (endothelioma).	"
12	30238	Enlarged vein from region of tumor...	Postcentral (osteosarcoma)	"
	30263			
13	27165	Dilatation meningeals, slight bilateral.	Hemisphere (cyst arach.)..	"
14	27982	" " " "	Temporal region (gumma).	"
15	27975	General vascular enlargem'nt, bilateral	Precentral (glioma).....	"
16	29878	Enlargement meningeal A, bilateral...	Hemisphere (subcort. gl.)..	Autopsy.
	30039			
17	29329	Dilatation meningeals slight, bilateral.	Frontal (endothelioma)...	Operation.
18	35766	" " " "	Temporal lobe (glioma)....	Operation; autopsy.
19	35952	Enlarged vein from region of tumor, unilateral.	Paracentral (endothelioma).	Operation.
20	15966	General vascular enlargement, slight..	Hypophysis (adenoma).....	"
21	23254	Stellate diploetic veins, parietal region	" (cyst).....	Operation; autopsy.
	26435			
22	33036	" " " " "	" (cyst).....	Operation.
23	35312	General vascular enlargement, slight..	" (adenoma).....	Operation; autopsy.
24	35387	Enlargement meningeal A, slight.....	Suprasellar, sarc. chiasm..	Operation.
25	21779	General enlargement, stellate diploetic veins.	Cerebellum (cyst).....	"
26	25057	Enlargement meningeal A, slight.....	" "	"
27	33072	" veins over occiput.....	Cerebellopontine (endothelioma).	"
28	32428	" meningeal A, bilat. slight	Pons (aneurism, basil. art.)	Autopsy.
29	31250	General vascular enlargement.....	Cerebellopontine (endothelioma).	Operation; autopsy.
30	17519	Enlargement meningeal A, bilat. slight	Presumed cerebral.....	No.
31	17306	" " " " "	" "	"
32	20112	" " " " slight, stellate diploetic veins.	" "	"
33	20717	Enlargement meningeal A, bilat. slight	" "	"
34	21035	" veins over frontal region..	" "	"
35	15214	" meningeal A, bilat. slight.	" "	"
36	20650	" " " " "	" "	"
37	16590	" vessels over occiput.....	" "	"
38	28790	General vascular enlargement.....	" "	"
39	27017	Enlargement meningeal A, bilat. slight.	" "	"
40	28733	" " " " "	" "	"
41	30777	General enlargement slight, stellate diploetic veins.	" "	"
42	30365	General vascular enlargement, slight..	" "	"
43	34451	Enlargem't meningeal A, bilat. marked	" "	"
	34610			
44	21728	General enlargement, stellate diploetic veins.	" Cerebellar.....	"
45	15280	General enlargement, slight, bilateral.	" "	"
46	15478	Enlargement vessels over frontal region	" "	"
47	17842	Stellate diploetic veins, parietal.....	" "	"
48	18037	Enlargem't meningeal A, bilat. marked	" "	"
49	17077	" " " " slight.	" "	"

A superficial tumor, a local increase in the number and size of the blood vessels of the adjacent dura and bone. In the roentgenograms of 50 patients we have noted an enlargement of the cerebral vessels, either a dilatation of the middle meningeal arteries or of the diploetic or extracranial sinuses of the skull. In those of 46 patients the dilatation is general and often of slight grade—an indication of increased intracranial pressure, but of no value in the focal diagnosis of brain tumor. It is seen most commonly in patients with cerebral or presumed cerebral lesions; more rarely in those with posterior fossa tumors. In the roentgenograms of four patients there is, as demonstrated with the stereoscope, a marked, in one, an enormous, *unilateral* dilatation of the cranial vessels—a finding of great value in the focal diagnosis of tumor. The roentgenograms clearly show a circumscribed network of vessels which drain into a single large vascular trunk. In all four patients the lesion at operation proved to be a dural endothelioma lying within the area outlined by the vascular network. In one, without other localizing signs, the unilateral vascular dilatation enabled us to make a positive focal diagnosis.

A stellate arrangement of the diploetic sinuses in the parietal region of the skull has been observed in the roentgenograms of eight patients. It occurs apparently regardless of the location of the lesion, and in our experience is merely a sign of venous stasis.

SUMMARY.

1. With the exception of the comparatively few which show definite tumor-shadows, roentgenograms of the head are merely an aid, though an important aid, in the diagnosis of brain tumor.

2. Uncalcified tumors do not cast shadows in the roentgenogram, unless tumor-tissue has invaded the accessory sinuses. A possible exception may be hypophyseal lesions which are viewed against the dark temporal fossa.

3. Calcified or bony tumors cast shadows which are readily recognized. In our experience such shadows occur in 6 per cent of patients with brain tumor. Judging from the literature, however, our experience has been fortunate.

4. The signs in the skull of increased intracranial tension, *i. e.*, enlargement of the skull, separation of the cranial sutures, general convolutional atrophy and destruction of the sella turcica, have a considerable value in the differentiation between cerebral and subtentorial lesions; for they indicate an internal hydrocephalus, which in our experience occurs only rarely in cerebral tumors, but is the usual accompaniment of posterior fossa tumors. It is of importance to remember that destruction of the sella turcica may be a general pressure phenomenon, especially in the differential diagnosis between suprasellar and cerebellar tumor in blind patients.

5. The local changes in the skull due to brain tumor are, in our experience, of greatest value in the diagnosis of hypo-

physeal or suprasellar lesions. The combination of characteristic eye-changes and local sellar destruction or enlargement makes the diagnosis the most certain, perhaps, of all intracranial conditions.

6. Local hypertrophy of the skull over cerebral tumors is of definite diagnostic value and has occurred in 4 per cent of our patients. Local atrophy of the skull over tumors is of equal diagnostic importance, but has occurred in only 2 per cent of the patients. Local unilateral vascular changes also have definite diagnostic significance and have occurred in 4 per cent of the patients. Local convolutional atrophy is of importance in the focal diagnosis of tumor only when demonstrably unilateral; in our experience this is rare. Local enlargement of the internal auditory meatus has thus far in our experience had very little diagnostic value.

7. The usual position and characteristic appearance of shadows due to the calcification of structures normally present in the intracranial chamber should be remembered.

8. In about 45 per cent of the patients in this series roentgenography has been of real diagnostic value.

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EXPLANATION OF PLATES.

Two pictures are shown over each figure number. The upper picture is the reproduction of a print made directly from the X-ray negative; the lower, a reproduction of a similar print retouched by an artist in order to emphasize abnormalities in the plate upon which we have commented in the text.

It should be remembered that the pictures represent positives of the X-ray negatives; and that, therefore, tumor-shadows, for example, which appear as dark shadows in the pictures appear as light areas in the X-ray plates.

CONTRIBUTION TO THE STUDY OF PROGNOSIS IN TUBERCULOSIS.[1]

By Herbert M. King, M. D.,

Loomis Sanatorium, Loomis, N. Y.

The present study is taken from a larger and more exhaustive inquiry into various phases of tuberculosis which we have had in progress at Loomis Sanatorium for the past few years, and which, I hope, will be ready for publication within the year. In the course of this inquiry we have arrived at certain results which, when considered in connection with the statistical studies that have been made previously along similar lines—notably those of Brown and Pope[2] and Noel Bardswell[3]—seem to justify some general conclusions as to prognosis, and in addition to suggest a more definite objective in treatment than has hitherto been emphasized.

I am not unmindful of the fallacy of drawing broad conclusions as to prognosis from the comparatively small numbers of cases so far dealt with. However, even though the numbers considered in any one of the available published reports be comparatively small, it does not follow that larger groups would show marked variation as to results, provided similar classifications were employed. Moreover, when approximately similar results are reached by different observers in the analyses of several relatively small groups, they would seem to have the weight of like results from a single analysis of a much larger group.

[1] Read before a meeting of the Laennec Society, May 5, 1916.

[2] Lawrason Brown and E. G. Pope: The Ultimate Test of Sanatorium Treatment. Zeitschrift für Tuberkulose, 1908, XII.

[3] Noel Dean Bardswell: Treatment of Pulmonary Tuberculosis. Lancet (London), March 8 and March 15, 1913.

In so far, therefore, as the figures here presented approximate those reached by others in similar inquiries, we may safely accept them as indicating the trend, at least, which would develop in the analysis of like groups regardless of numbers.

The material here considered consists of 1,515 cases of tuberculosis, all except seven of which were pulmonary. All the patients were under observation and treatment in Loomis Sanatorium for at least one month (the average stay for the various classifications in different years being from six to nine months), scattered over the period from the opening of the sanatorium in 1896 to October 31, 1910. These cases were variously classified upon admission as follows: Incipient, 256; moderately advanced, 634; far advanced, 618; and seven with lesions elsewhere than in the lungs. The classification schema is the simple form of that of the National Association for the Study and Prevention of Tuberculosis.

This group of 1,515 patients was selected because, of the individuals comprising it, we have the following definite information: (a) We know that all were living at the time of discharge; (b) we know whether they were living or dead at the expiration of five years thereafter; (c) of the dead, we know at what period within the five years death occurred; (d) we know whether or not *B. tub.* was demonstrable in the sputa of these patients while they were in the sanatorium.

Much other information we have respecting this group, but for present purposes these four points suffice, and by limiting the inquiry we avoid that attenuation of numbers which further division would entail, and which, in the consideration of so small a group, would necessarily render deductions somewhat uncertain. There can be no ambiguity in the answers to these four questions. If, on the other hand, we attempt to subdivide the living into "Health satisfactory" and "Health unsatisfactory," we are dealing largely with patients' opinions, which may or may not be correct; but "Alive" or "Dead" admits of no qualifications.

Limiting the inquiry to a period of five years is perhaps more arbitrary. We know that many patients with chronic tuberculosis are alive five years after discharge from the sanatorium, notwithstanding that their disease is progressive, and that quiescence has never been established, nor is likely to be. But the advantages of choosing such a comparatively short period for such a study as this are several: (a) It suffices to indicate the trend through the most critical period; (b) it is the maximum period which would apply to all cases alike in this particular group; (c) it eliminates to some extent the element of an increasing death rate, from causes other than tuberculosis, incident to advancing age; and (d) it serves to convey more easily and graphically a mental picture of the life or death curves, and is therefore more practicable for an occasion like the present.

If we can estimate with any degree of accuracy the chances of life among the patients now under consideration for five years subsequent to their discharge from close observation and treatment, we have gone far toward establishing an ultimate prognosis; for, as will be seen by reference to the charts, the death rate tends to become more nearly constant after the third year and probably would be still more so if the group comprised thousands instead of hundreds.

There is no intention in this brief study to argue for the sanatorium or any other method of treatment. Many of the group, especially among the far advanced class, had been under treatment, either at home or in resorts, both before and after their sanatorium experience.

The object here is rather to inquire what may reasonably be expected from intelligent care and treatment in various classes of tuberculous invalids, with the assumption, to start with, that a patient who will spend at least a month (in most instances many months) in a sanatorium will avail himself of every means of recovery within his reach, and with his sanatorium training will avoid, at least, the more dangerous pitfalls which beset the consumptive.

Proceeding then to an examination of the charts: Nos. 1, 2 and 3 are arranged to indicate in graphic curves the deaths among various classes of tuberculous patients during five years after discharge as compared with those among the general population; and Chart 4 is a table showing the chances for or against life during the five years among these various classes. In all the charts the various classes are considered on a basis of 1000 in order to facilitate comparison with the accepted figures for the general population, as well as with each other. The actual number of tuberculous individuals, however, is indicated at the termination of the curves in each of the graphic charts.

CHART 4.

COMPARISON OF ODDS FOR OR AGAINST PROBABILITY OF LIFE AT THE END OF FIVE YEARS.

	Number in each one thousand alive at end of five yrs.	Number in each one thousand dead at end of five yrs.	Odds for or against the probability of life at the end of five years.
General population	951	49	19.4 to 1 in favor.
Tuberculous patients by condition on admission.			
Incipient	859	141	6.1 to 1 in favor.
Moderately advanced	558	442	1.3 to 1 in favor.
Far advanced	222	778	3.5 to 1 against.
Tuberculous patients by condition on discharge.			
Apparently cured	919	81	11.3 to 1 in favor.
Arrested	662	338	1.9 to 1 in favor.
Improved	385	615	1.6 to 1 against.
Unimproved	50	950	19.0 to 1 against.
Tuberculous patients.			
No bacilli demonstrable during residence.	874	126	6.9 to 1 in favor.
Sputum changing from bacillary to non-bacillary	773	227	3.4 to 1 in favor.
Sputum bacillary on discharge.	287	713	2.5 to 1 against.
All classes of tuberculous patients combined.	473	527	1.1 to 1 against.
Patients discharged with disease quiescent but with bacillary sputum.	607	393	1.5 to 1 in favor.

Chart 1 is plotted to show the death curves among patients classified as to their condition upon admission without reference to their condition upon discharge. It presents, graphi-

cally, the usual argument in favor of placing the patient under treatment at the earliest possible stage of the disease. It illustrates what might be expected with regard to far advanced cases and, to a lesser extent, the moderately advanced group, *i. e.*, the high death rate during the first two years after discharge, with a subsequent tendency on the part of the curve toward the horizontal. In this respect it corresponds, though in far less degree, with the curves of the improved and unimproved classes on Chart 2. These two charts, however, should not be compared one with the other, since they are dealing with such different groupings.

It will be noticed that the death rate in the incipient class, in contradistinction to the two other groups, increases after the second year, and the curve does not again trend toward the horizontal until the last year of the period. Although this group is so small, this peculiarity of the curve is probably not accidental, but is quite what might be expected. The progressive cases of the group classified as incipient naturally have a longer interval before the disease reaches a fatal termination than is the case with the more advanced classes. From a comparison of these curves it is possible to estimate roughly the relative length of life among those individuals of the several groups in whom care and treatment fail to arrest the disease. Turning now to Chart 4, we have, expressed in proportion, the chances for or against life five years after discharge from treatment, as applying to this grouping.

It must be borne in mind, however, that the mathematics of chance cannot be strictly applied here, but only in such measure as to furnish a reasonably safe basis for a broad comparison. For instance, in the far advanced class of tuberculous patients, the odds figure 3.5 to 1 against the chance of life at the end of five years; but this group is made up of individuals who were discharged, some apparently cured, some arrested, some improved and some unimproved. Obviously, it cannot be said that these odds will apply in general to the far advanced stage irrespective of condition on discharge; so that the figures are of value only as a means of comparing the far advanced group with other groups to which the same or similar restrictions apply. Nevertheless, it is probable that were the same grouping made from other reliable records and the same schema of classification employed, the results would approximate these fairly closely.

The variation in chances, as here shown, between the incipient and the far advanced classes is wide—6.1 to 1 in favor of life in the former as against 3.5 to 1 against life in the latter. These figures, as respecting the incipient class, I think will be found approximately correct and fairly safe for general application; but in the case of the far advanced, with our present crude schema of classification and the wide variation in condition of individuals that our present schema necessarily throws into this class, one would have to be much more guarded in expressing relative chances mathematically.

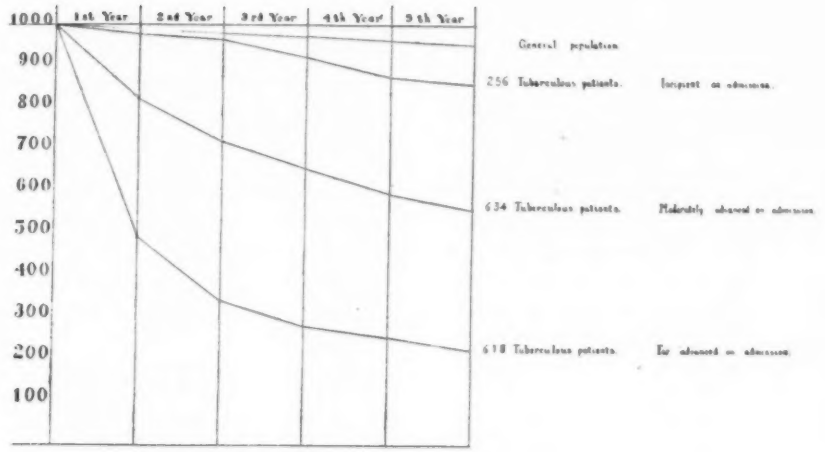
Chart 2, as you see, illustrates in graphic curves the history, for five years subsequent to discharge, of the same group of patients, but classified as to condition on discharge without reference to condition upon admission. In this chart it is noticeable that the death curve in the apparently cured class differs very slightly from that of the general population—indeed, is parallel for the first year—and later diverges very gradually. If this class were made up exclusively of patients classified upon admission as incipient, the curve would approximate that of the general population even more closely, as naturally the "apparent cures" among the advanced classes are more prone to relapse. The curve as applied to the unimproved is spectacular, not to say shocking—76% dying within the year after discharge, and only 5% remaining alive after five years. These curves, I think, would be found to apply generally were similar classifications employed with other reliable data, and they serve to illustrate what may be expected for five years subsequent to discharge from modern treatment in a similar group of patients.

To turn again to Chart 4 for an expression in figures of the chances for or against life within the period considered, we find that the apparently cured enjoy odds of 11.3 to 1 in favor of life, while among the unimproved the chances against living are 19 to 1, or practically the same probability of dying within the five years as the normal population has of living for the same period. This chart argues one point which I think is worth remark, and that is the relative accuracy of our present plan of classification upon discharge. No schema of classification so far devised has proven wholly satisfactory; but, from a study of ultimate results, comparatively little fault can be found with this feature of our present schema.

So much for prognosis as indicated in a study of the comparatively limited group of patients here considered. The charts speak for themselves within the restrictions imposed by the somewhat small number of individuals and the limited period of time which we have chosen. Comparing the results with those of other observers who have published records of similar groups considered in like manner, there is no wide discrepancy, and in so far the curves here presented may, I think, be very generally applied.

In the beginning of this paper I stated that, in addition to the question of prognosis, certain results of our inquiry had suggested a more definite objective in treatment than had hitherto appeared as such in any of the published reports with which I am familiar. Let me very briefly explain:

The great majority of patients who, for the first time, come under treatment in a sanatorium or in the open resort have but the vaguest idea of what specific thing they have come there to accomplish—to recover from tuberculosis, of course; but when told that the term "cure" is not now employed; that it is impossible to say with definiteness when an "arrest" has been achieved; that certain adventitious physical signs are not incompatible with "economic health," so to speak; that slight cough and expectoration may persist through the remainder of their lives to remind them of their lesions, and that enormous weight-gain does not mean recovery in tuberculosis, it is quite natural, I think, for them to wonder just what criterion they are to employ to determine the length of their sacrifice and the period at which they may with greatest safety return to their normal occupations and home environment.



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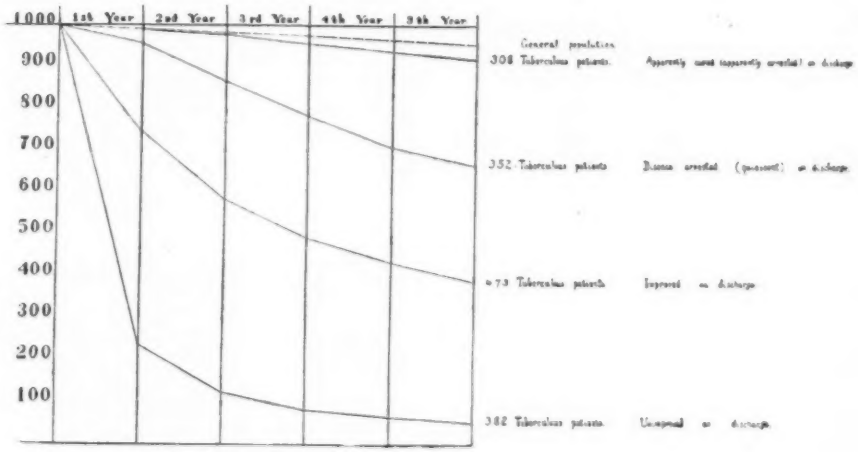

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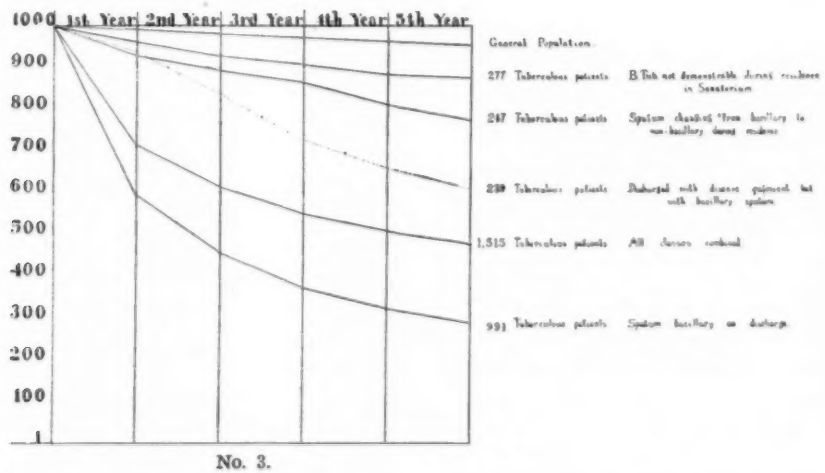

No. 3.

GRAPHIC CHARTS SHOWING DEATH CURVE OVER A PERIOD OF FIVE YEARS IN GENERAL POPULATION, AS COMPARED WITH THAT OF VARIOUS CLASSES OF TUBERCULOUS PATIENTS.

This has not been an easy question for the specialist to answer, much less for the family physician who has discovered the disease and sent the patient away. It may be somewhat disconcerting, but it is none the less a fact that comparatively few patients, outside of the incipient group, among the various classes going to make up the personnel of tuberculous communities, can or will remain under close observation and treatment long enough to become permanently arrested cases.

It therefore becomes almost necessary, nowadays, to hold out both to the patient and to the physician some definite objective, toward which the patient may look with some reasonable chance of achievement, as a goal the attainment of which will free him from the more irksome restrictions that surround him while under systematic treatment. In an article published some years ago by Noel Bardswell, which had to do with records of tuberculin-treated cases, attention was first drawn, so far as I am aware, to the importance of changing a lesion from an "open" to a "closed" one. This he considered the paramount and unequivocal sign of improvement. In the course of our inquiry at Loomis the great advantage of this attainment has been proved by the histories of such cases after discharge from the sanatorium; so that we have come to hold out this achievement as the most important single desideratum toward which the patient should direct his efforts, and for which he should exercise his patience and perseverance. It has the advantage of being definite; it is a question which can be answered categorically, and it seldom fails to appeal to the intelligence of the average patient, who will make the greater effort when he has a specific objective. Unfortunately, it is difficult of achievement in the more advanced cases within the limits of time which financial considerations usually permit a patient; but it comes more easily than does an apparent cure, and it is something which can be striven for and often attained after the patient has resumed his normal vocation and environment.

Chart 3 is plotted to illustrate the foregoing argument. It is made up of the same group from which the other charts were constructed, and is, like them, self-explanatory. The patients discharged as non-bacillary are, as will be noticed, separated into two sub-groups, which separation, while it attenuates the numbers, does away with the objection that might otherwise be raised regarding such cases as were never found to be bacillary. Bear in mind that, of the sub-group changing from bacillary to non-bacillary, there can be no ambiguity as to diagnosis, and that many in this division were discharged with no better classification than "arrested"—some merely "improved" and a few "unimproved." With these points in consideration, a comparison of the several curves shows unmistakably the advantage in prognosis which the loss of bacilli gives a patient. Obviously, it would not be fair to compare the death rate in the non-bacillary class or in the class losing bacilli during treatment with the class discharged "bacillary," since the latter would include all of the progressive cases discharged "unimproved," in which, as we have seen, the prognosis is very bad. In order, therefore, that we may have two approximately comparable groups, another sub-group has been formed. It is indicated on the chart by a dotted line and comprises patients all of whom were discharged with disease "arrested," but with bacillary sputum; while in the group with which it is to be compared, all of whom were discharged without bacilli, there are unavoidably included a few patients who left the sanatorium only "improved" and some "unimproved"—a small, unfavorable element which accounts for the apparently anomalous and slightly greater death rate in the non-bacillary class during the first year as compared with that in the "arrested" bacillary group. The "improved" and "unimproved" in the non-bacillary group would naturally be nearly all eliminated during the first year.

A comparison, now, of the curves under discussion is, I think, very convincing of the advantage of becoming non-bacillary, and seems to justify the contention that, short of an "apparent cure" of the disease, with all that the term implies, the achievement of a "closed" lesion is the chief definite objective for which the patient and his physician should strive during the months of what might be termed the "active treatment of the disease."

For the interesting charts and statistical figures upon which this study is based I am indebted to our statistician, Mr. Cornelius W. Dever and to Mr. Raymond P. Woodman of the Loomis staff.

DISCUSSION.

DR. LOUIS HAMMAN: I have listened with much interest to Dr. King's presentation. On a number of occasions I have gone over reports upon large numbers of tuberculous patients and I appreciate fully the value of a study of this kind made upon patients who have been observed and classified in a uniform way. Most statistical studies of tuberculosis handle material gathered from various sources and arranged and judged by different standards. Large figures are imposing, but one familiar with the methods of observing and classifying patients will at once appreciate the possibility of grave error. Statistical studies comprise two parts, the medical data upon which the figures rest and the mathematical manipulation of these figures. The latter part is developed into an exact science, but all of this exactness is wasted if the medical data are inaccurate and unsatisfactory. Therefore, a small group of cases observed by one man, or at least in a single institution with permanent traditions, is of far greater value than elaborate mathematical computations of material gathered from various and varying sources.

I recall one occasion when I tried to gather convincing figures to demonstrate the value of our tuberculosis campaign and to discover those measures of the campaign that had given the largest benefits. It was interesting to find observers drawing different and often opposed conclusions from the same figures. And another occasion when my object was to reveal the value of tuberculin treatment. Although numerous studies were at hand, I was surprised to find upon close analysis that the most enthusiastic reports often dwindled to personal impressions. I was convinced by the investigation that the only standards of value were whether the patients were living or dead at the end of a certain period, and the disappearance of tubercle bacilli from the sputum. I am pleased to note that Dr. King also has emphasized these two standards.

Frequently in discussion before this society I have called attention to the difficulties surrounding the diagnosis of early or incipient pulmonary tuberculosis. The diagnosis of incipient tuberculosis for purposes of treatment and for purposes of sta-

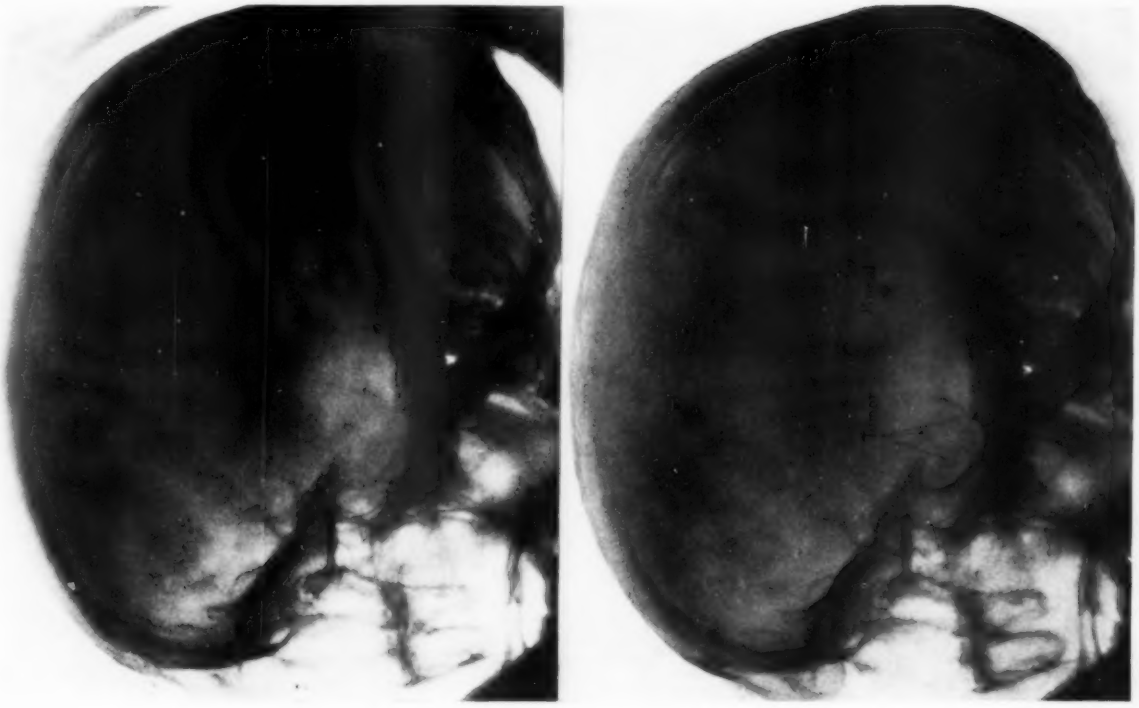

FIG. 2.—Roentgenogram of a patient with a pituitary tumor. The sella turcica is enlarged, the posterior clinoid processes partially destroyed. Within the sella a definite shadow cast by the tumor is plainly visible.

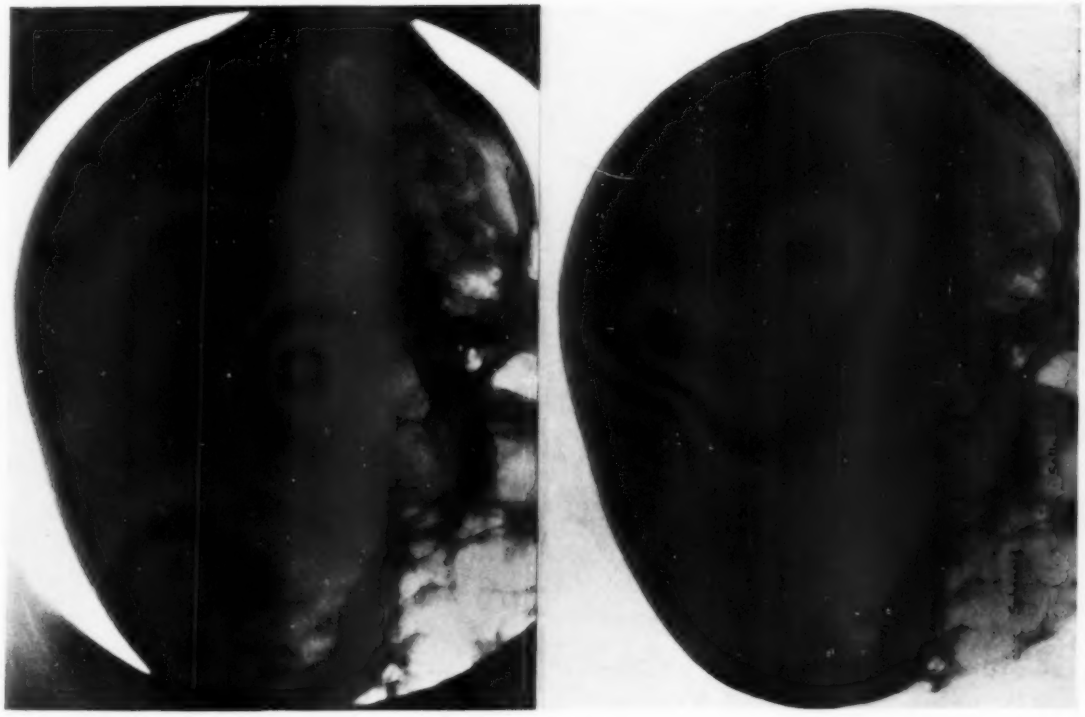

FIG. 1.—Roentgenogram of a patient with a pituitary tumor. The floor of the sella turcica shows a defect through which the tumor has entered the sphenoidal sinus. The shadow cast by the tumor-tissue clouds the sinus. The pineal gland casts a shadow.

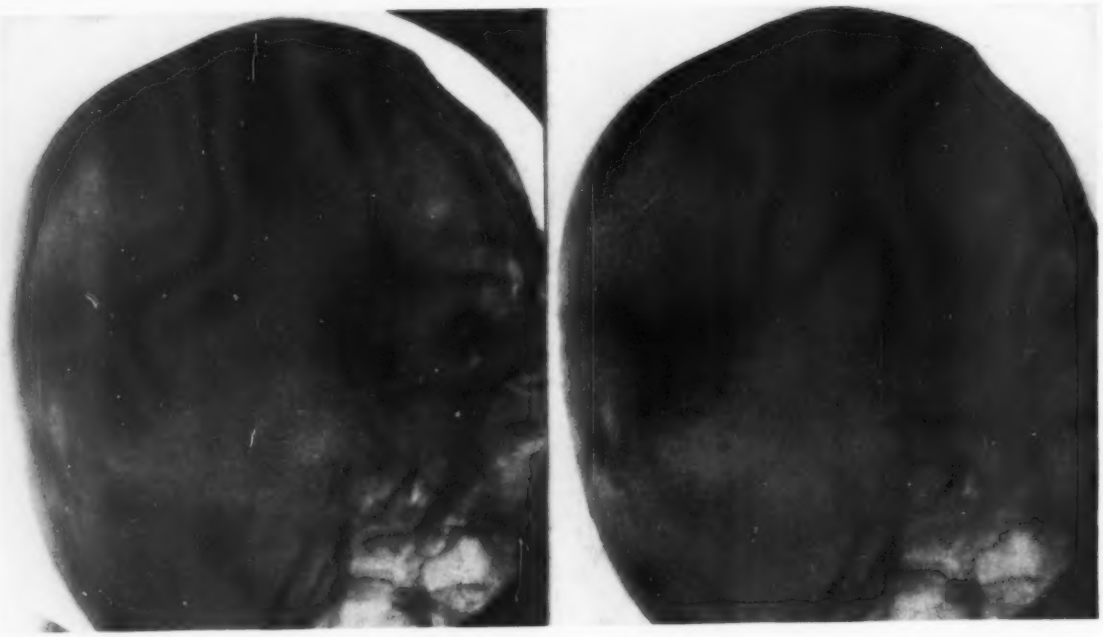

FIG. 4.—Roentgenogram showing the shadow of an osteosarcoma of the skull. Seen with the stereoscope, the tumor extends well into the intracranial chamber.

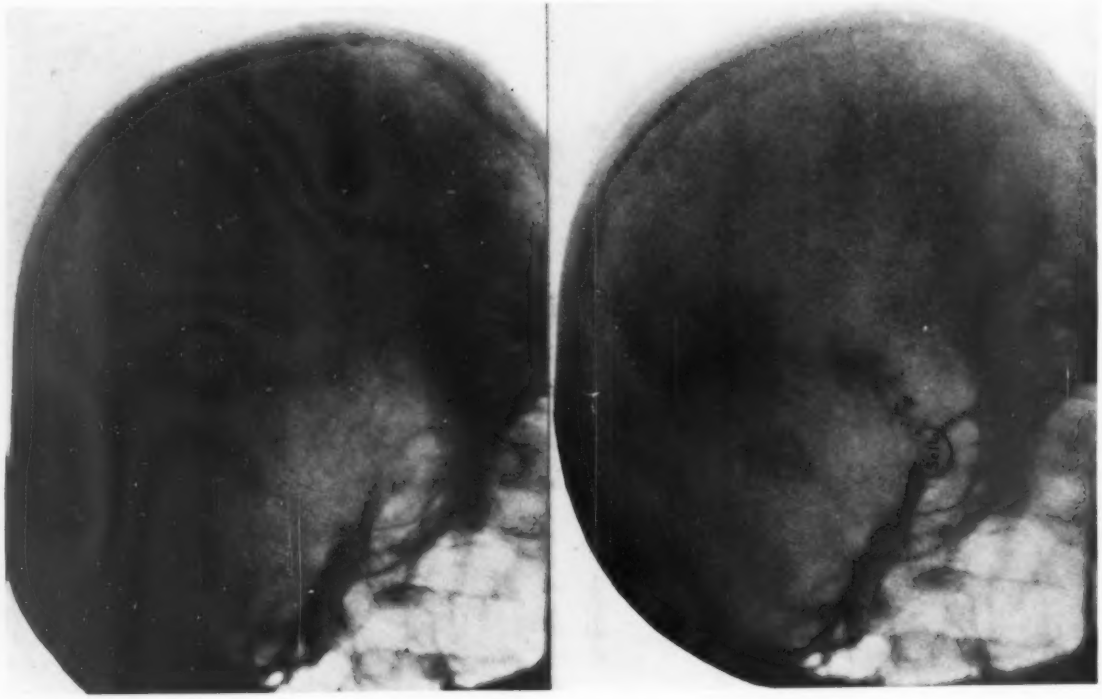

FIG. 3.—Roentgenogram showing the shadow of a calcified tumor. The tumor is probably a suprasellar growth which has extended upward into the hemisphere.

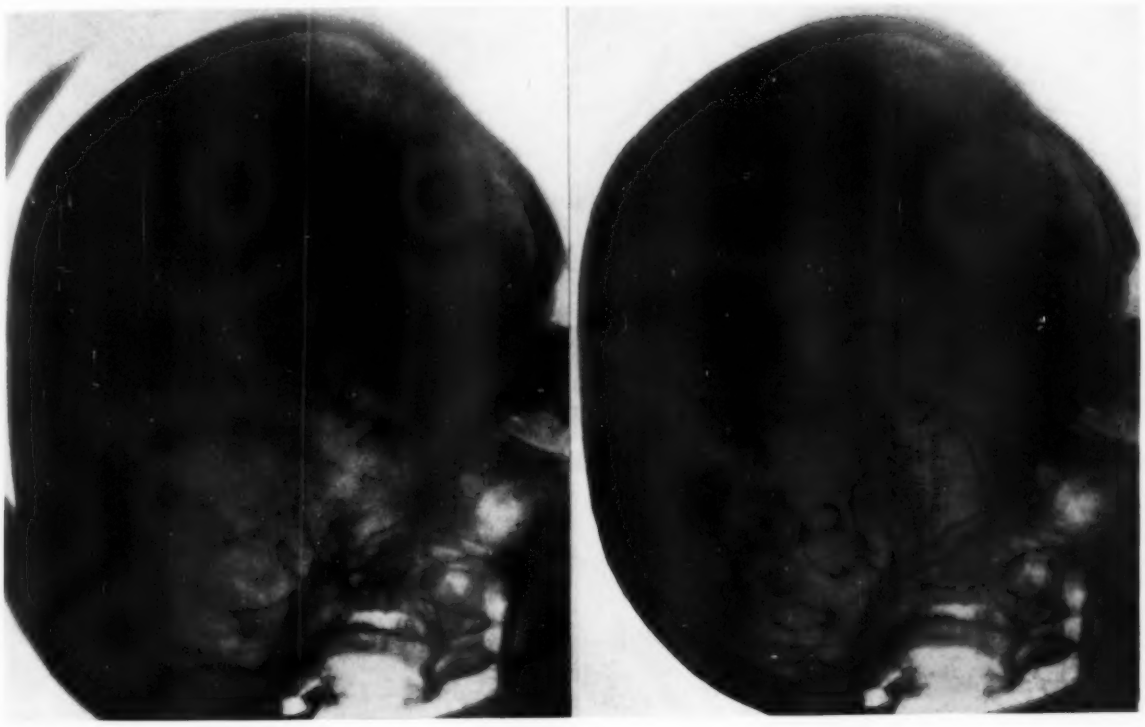

FIG. 5.—Roentgenogram showing the shadow cast by an aneurism of the internal carotid artery. The areas of calcification in the wall of the sac appear as dark, heavy lines. The sella turcica appears to be completely destroyed. The local convolutional atrophy of the inner table is seen in the frontal region.

FIG. 4A.—Specimen of osteosarcoma of the skull removed at operation. The bone and dural flaps were removed with the tumor. The roentgenographic shadow cast by this tumor is seen in Fig. 4.

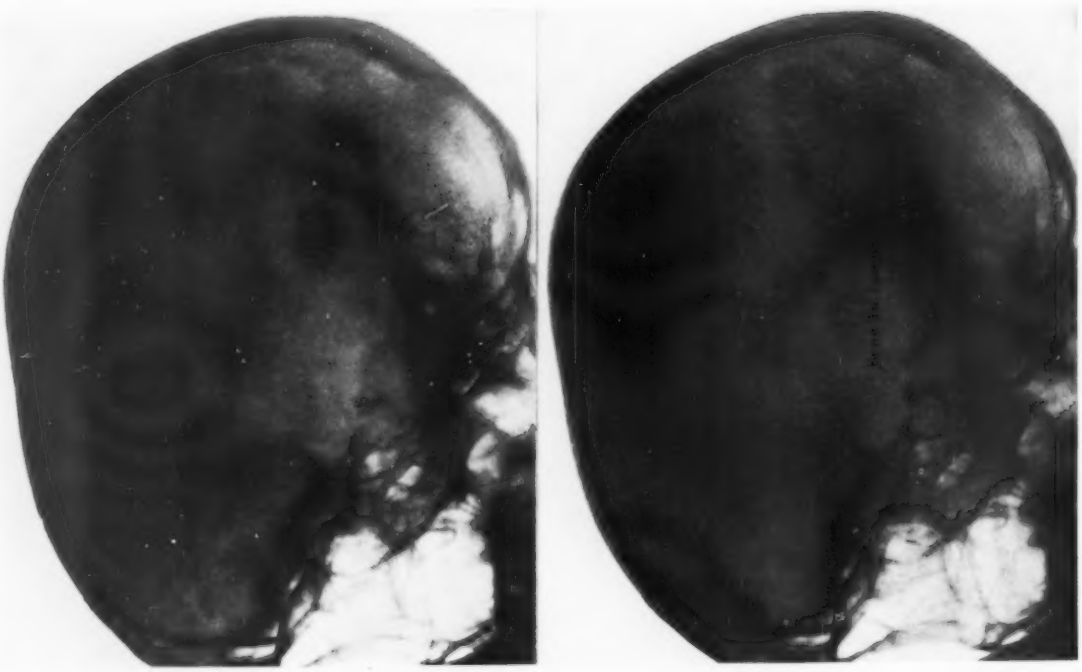

FIG. 6.—Roentgenogram of a patient with a suprasellar lesion (cyst of Rathke's pouch). The small shadow lying just above the posterior clinoid processes of the sella turcica is cast by an area of calcification in the wall of the cyst. Verified by autopsy studies.

FIG. 5A.—Specimen of aneurism of the internal carotid artery. The roentgenographic shadow cast by this aneurism is seen in Fig. 5.

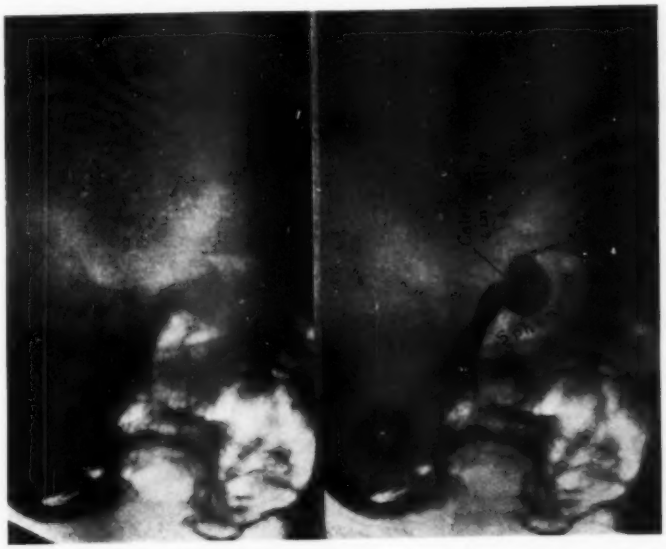

FIG. 8.—Roentgenogram showing the shadow cast by a calcified hypophyseal tumor. The shadow is so dense that the clinoid processes are obscured.

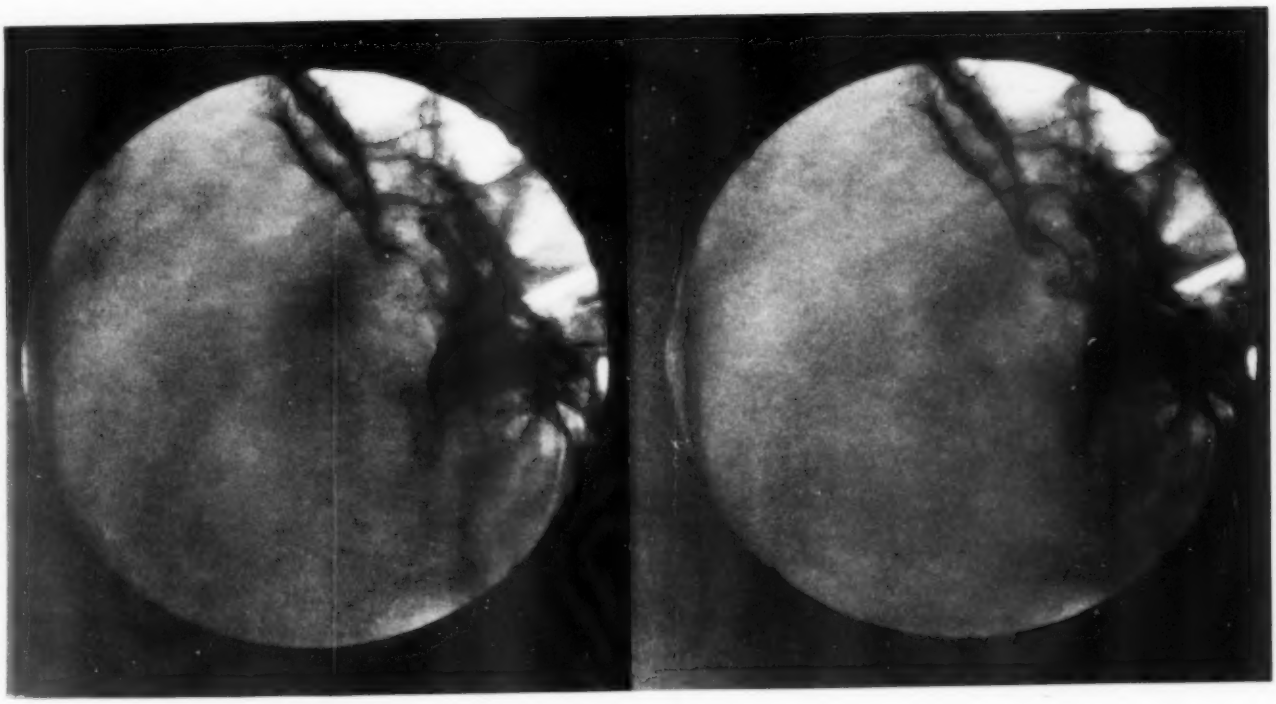

FIG. 7.—Roentgenogram of a patient with sarcoma of the optic nerve sheath. The small shadow appearing as a continuation upward of the posterior clinoid processes is an area of calcification in the tumor. The sella turcica is flattened.

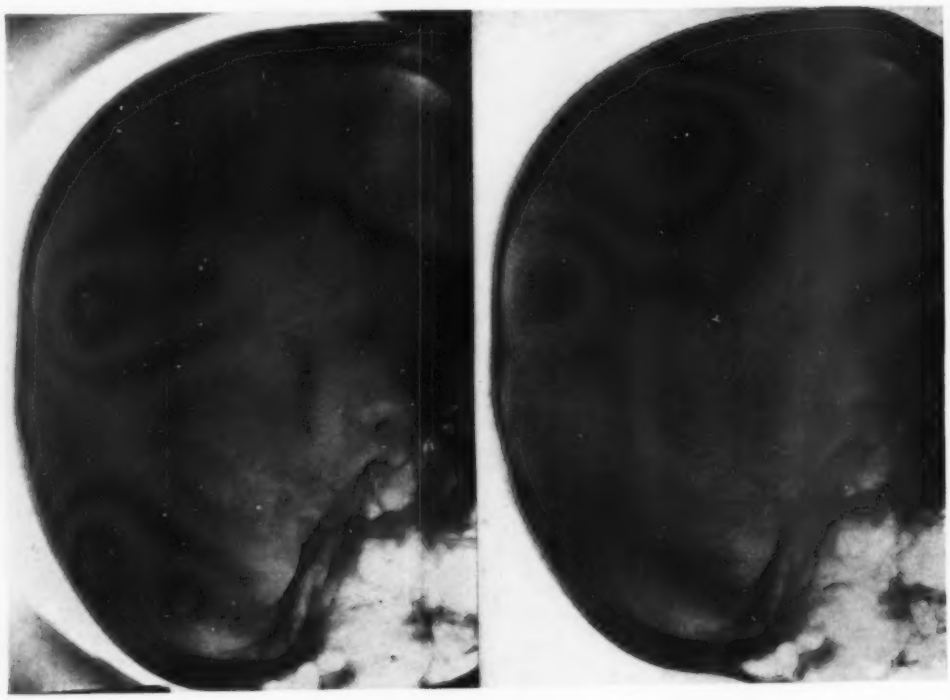

FIG. 10.—Roentgenogram showing the shadow cast by an area of calcification in the pineal gland.

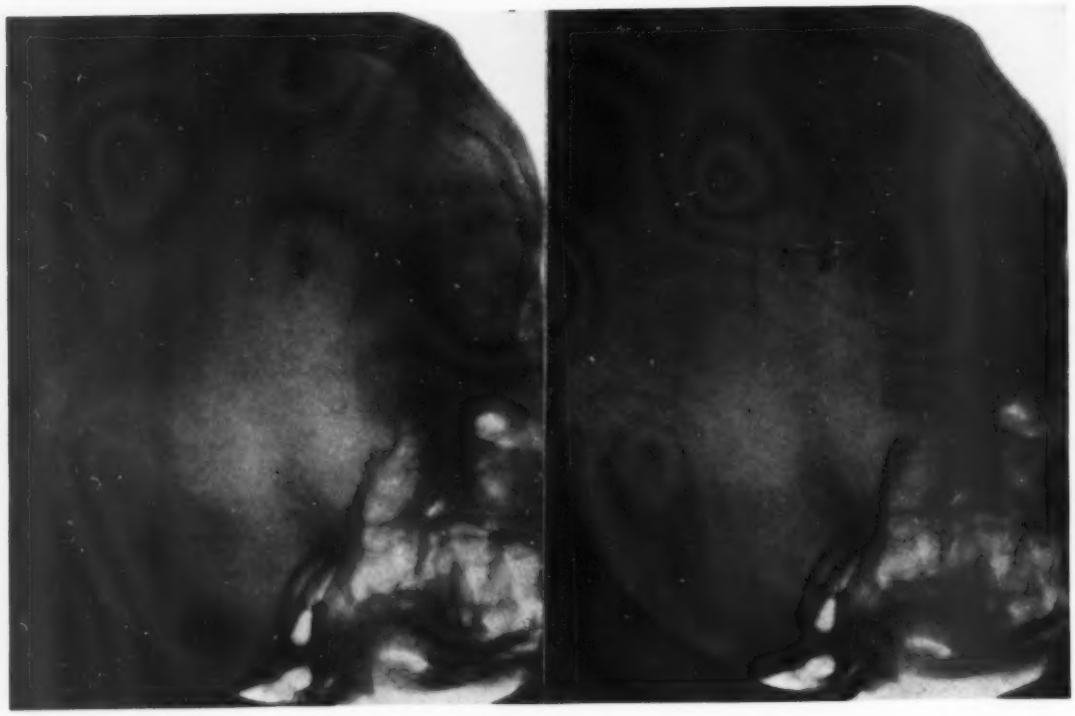

FIG. 9.—Roentgenogram showing shadows cast by areas of calcification in the choroid plexus.

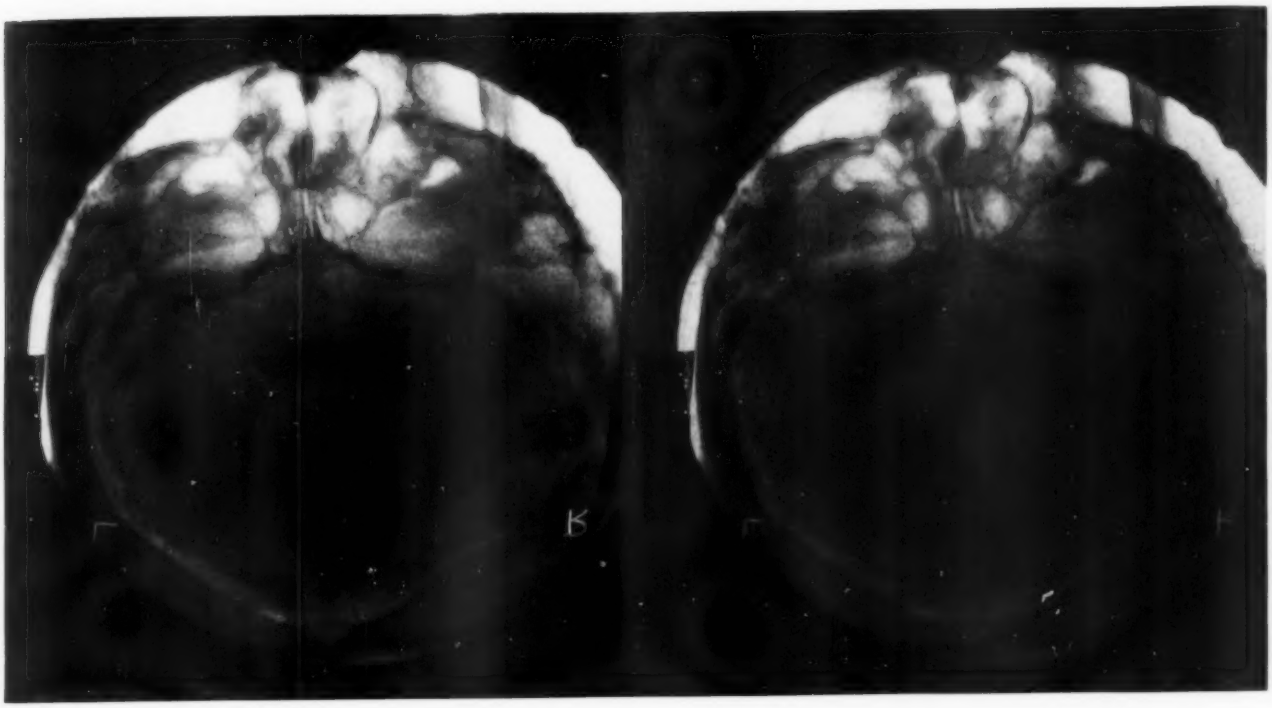

FIG. 11A.—Antero-posterior roentgenogram showing the shadow cast by an area of calcification in the falx cerebri.

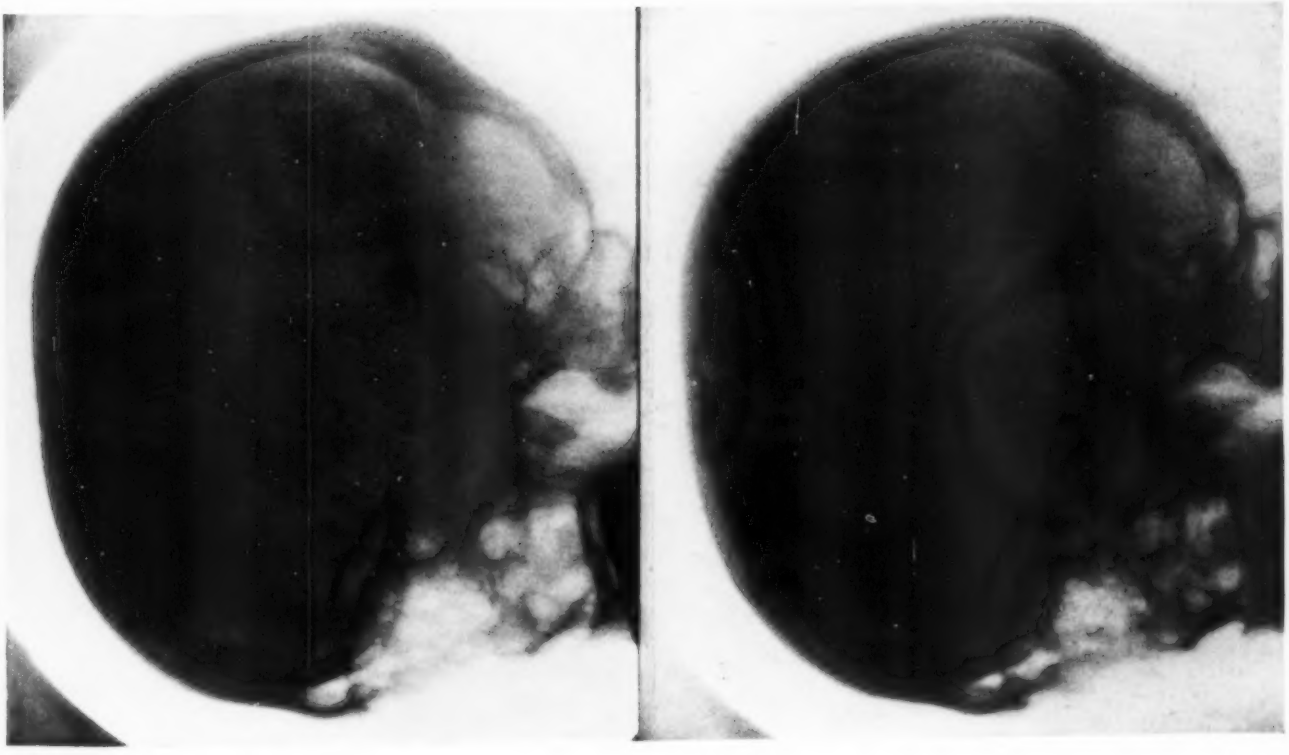

FIG. 11.—Roentgenogram showing the shadow cast by an area of calcification in the falx cerebri. The shadow might be mistaken for a tumor-shadow. Its position in the median plane is seen in Fig. 11a.

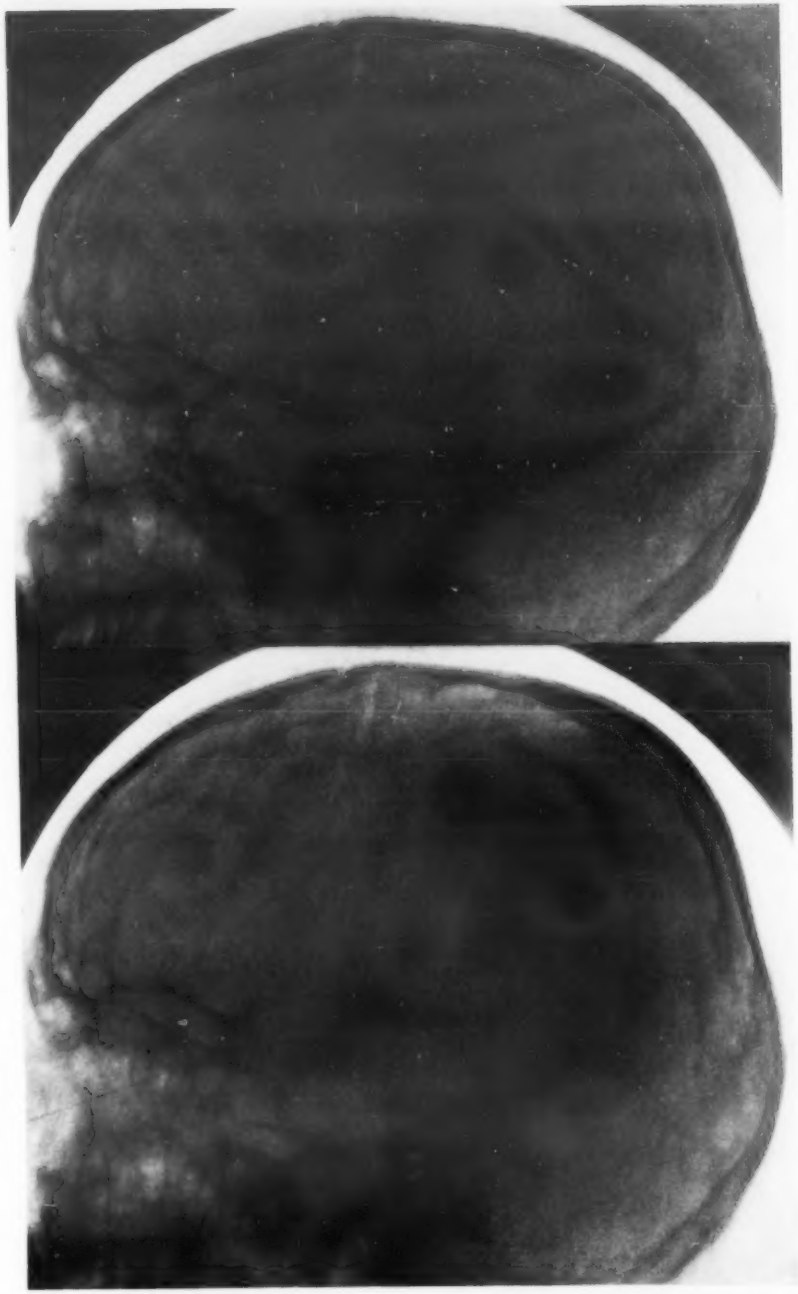

FIG. 12.—Roentgenogram of a patient with cerebellar tumor, showing enlargement of the skull, separation of the cranial sutures, general convolutional atrophy, and partial destruction of the sella turcica. The enlargement of the internal auditory meatus is evident.

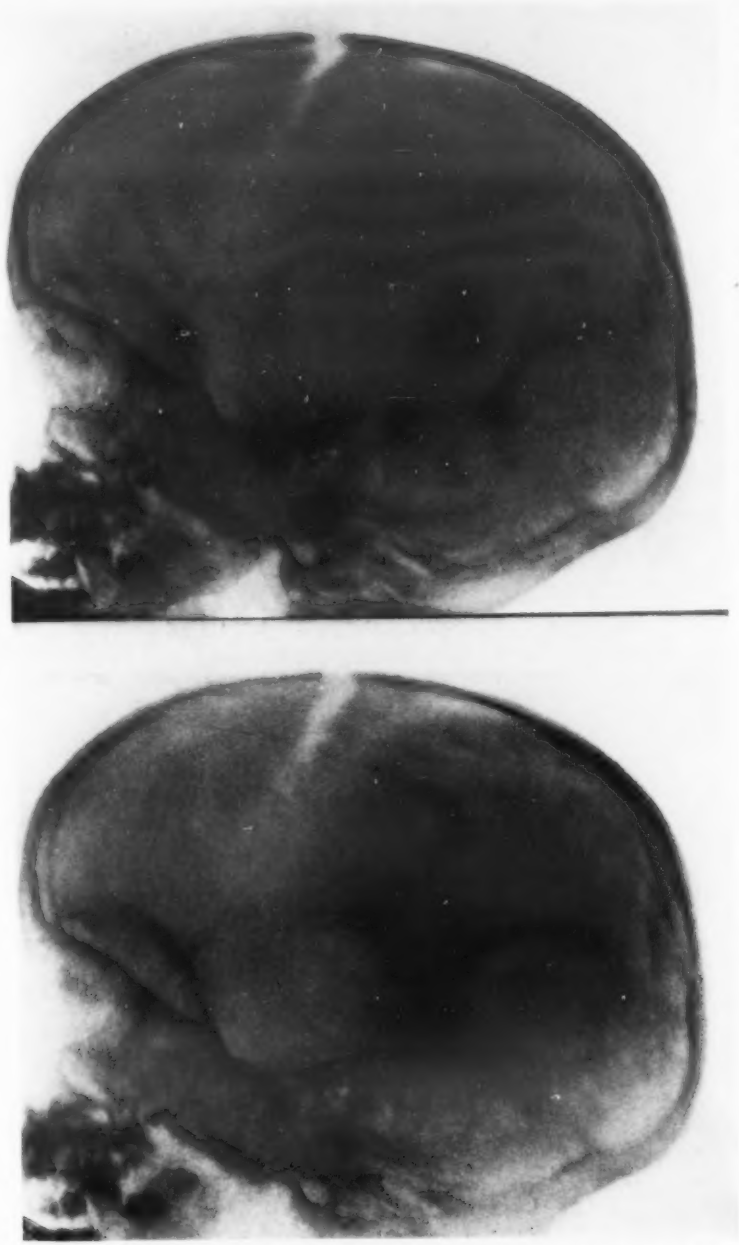

FIG. 13.—Roentgenogram of a patient with cerebellar tumor showing enlargement of the head, wide separation of the sutures, and complete destruction of the sella turcica. There is no convolutional atrophy. The internal auditory meatus is enlarged.

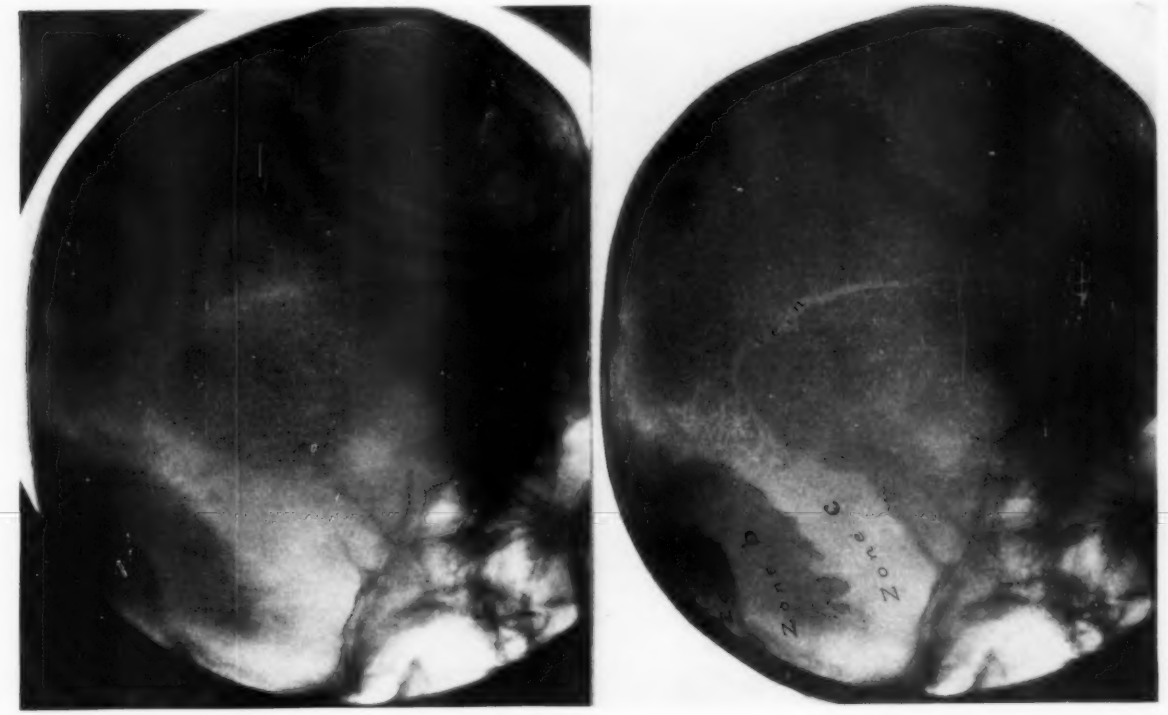

FIG. 15.—Roentgenogram of a patient with a dural endothelioma in the frontal region, showing a marked hypertrophy of the skull over the tumor (zones *a* and *b*). There is atrophy of the skull about the area of hypertrophy (zone *c*). Note the single large vein with its branches.

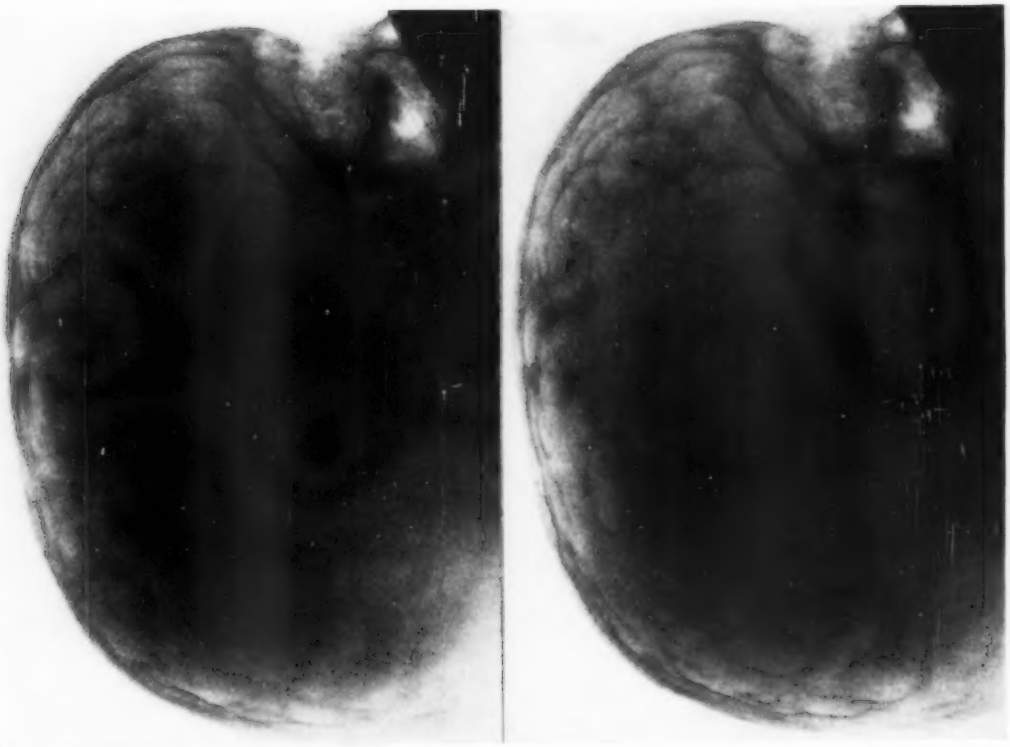

FIG. 14.—Roentgenogram of a child with "idiopathic" internal hydrocephalus, showing a general convolutional atrophy of the inner table of the skull.

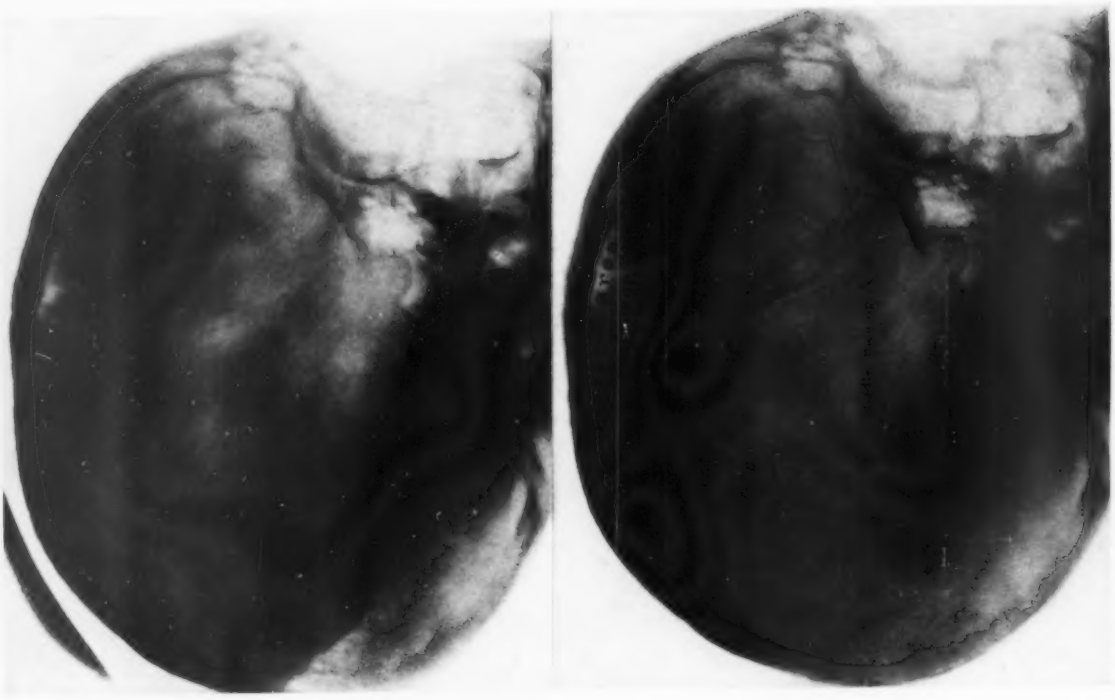

FIG. 17.—Roentgenogram of a patient with a dural endothelioma showing a local atrophy of the vault over the tumor. Note again the large vein (unilateral) coming from the region of the growth.

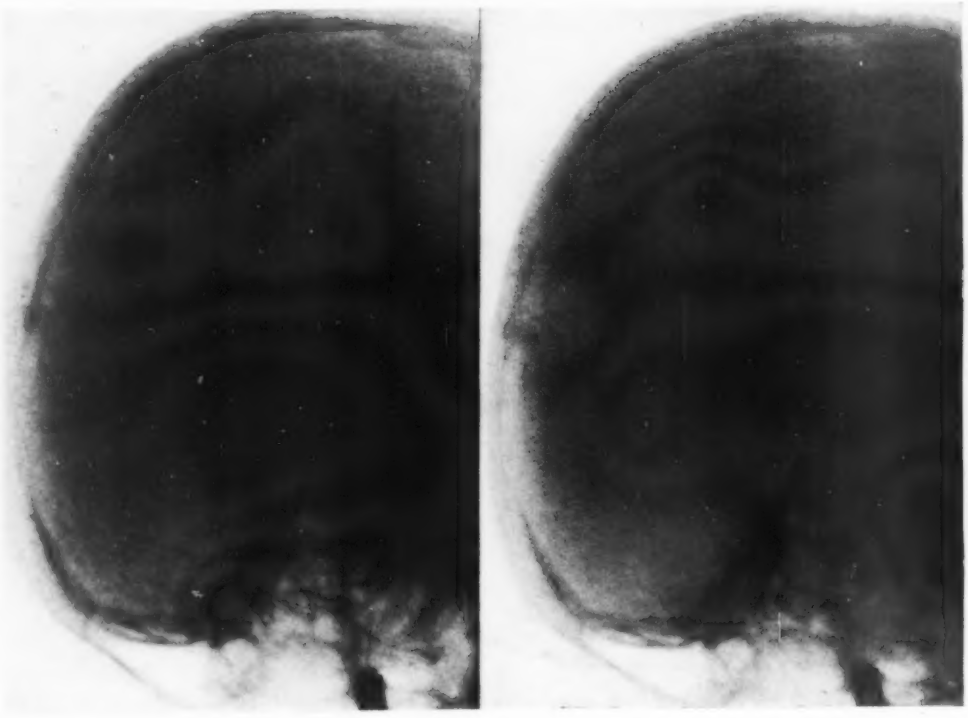

FIG. 16.—Roentgenogram of a patient with acromegaly, showing the local enlargement of the sella turcica without destructive changes and without alteration of the distance relations between anterior and posterior clinoid processes.

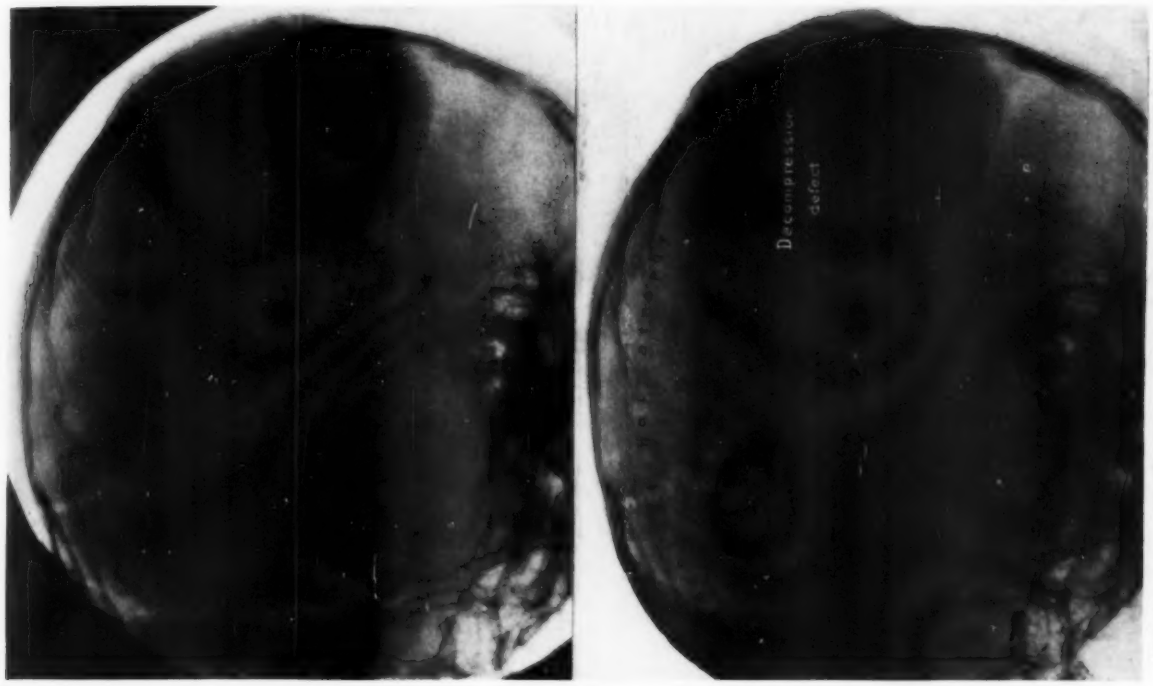

FIG. 19.—Roentgenogram of a patient with a suprasellar tumor showing complete destruction of the sella turcica. Note the signs of increased intracranial pressure, *i. e.*, convolutional atrophy and separation of the sutures.

FIG. 18.—Roentgenogram of a patient with a pituitary tumor showing the local enlargement and partial destruction of the sella turcica.

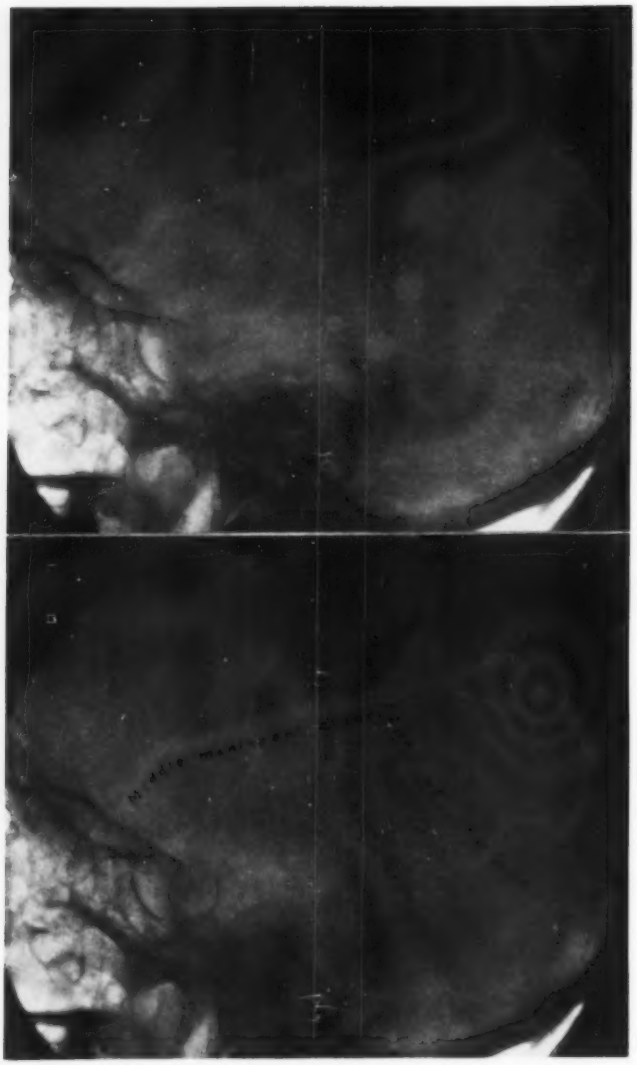

FIG. 20.—Roentgenogram of a patient with a dural endothelioma showing a marked unilateral enlargement of the meningeal artery. The outline of the tumor as found at operation is shown by the dotted line in the lower figure.

tistical study is widely different. In the one instance we are concerned with the safety of an individual; in the other our object is to ascertain the truth shorn of personal considerations. Standards of diagnosis do vary and always will in such an uncertain field. I need merely mention the difficulty of distinguishing active from inactive lesions. To be absolutely reliable statistical studies of pulmonary tuberculosis should include only patients with tubercle bacilli in the sputum, and since so small a proportion of patients with bacillary sputum are in the incipient group it follows that at the present time a satisfactory statistical study of the incidence and course of incipient pulmonary tuberculosis is beyond our reach. I am quite sure it is beyond our reach, and I am also sure that some of our impressions about this group are so strongly flavored with the personal equation that they savor of error. The strikingly favorable course of Dr. King's incipient cases raises the question about the diagnosis in this group. I know Dr. King will at once admit the possibility of error and at the same time insist, as I do, upon the impossibility of doing any better at the present time.

In the end prognosis must aim to be individual, that is, not only to tell the chances of life in a group, but to say what will happen to a particular patient. This is the ultimate refinement of the art towards which we strive. In the meantime, such careful studies as Dr. King has made carry us slowly towards the goal.

DR. H. B. JACOBS: I am struck with the value of these charts for use as tuberculosis propaganda. It is so difficult to get patients early enough. They do not appreciate the tremendous importance of losing no time in the beginning. If we could have such charts as these published to bring to the attention of those who are having an unremitting cough, it seems to me we might have in them means of impressing such with the value of early treatment as has not been found before.

DR. H. M. KING: I quite agree that the ultimate object in prognosis is to apply it to the individual. "Group" prognosis, so to speak, while it has a very distinct value, falls short of the requirements. It does not satisfy to say that a certain percentage of such-and-such a group recovers. One should strive to recognize the factors which go to determine the prognosis in the individual and then to apply the knowledge to the individual. As has been said, prognosis is a science, only we have not treated it as such. A study like this is but a step toward placing it where it belongs. Much remains to do—further statistical inquiry and a great deal of laboratory investigation—before we may venture individual prognosis in tuberculosis with confidence or assurance; but I do think we are now proceeding along right lines.

As to the question which Dr. Hamman raises regarding diagnosis in the incipient class, I think it is true that not a few cases remain under observation and treatment for months, classified as positively tuberculous, in which the one unequivocal diagnostic factor is lacking, namely, the presence of *B. tub.* in some of the excretions; and among these cases doubtless there are some, perhaps many, which, if the truth were known, are not tuberculous at all. For this reason I thought it well to separate the group of cases in which *B. tub.* had at no time been demonstrated, a group which, as you see, comprises 277 cases, some of which I have no doubt never had tuberculosis.

THE RENAISSANCE OF UROLOGY.[1]

By HUGH HAMPTON YOUNG, M. D., Baltimore, Md.

I have chosen as my subject the Renaissance of Urology. In explanation I may say that urology, comprising as it does the diseases of the urinary tract, was perhaps the very first branch of medicine to emerge from the clouds of ignorance in prehistoric times. This probably came about from the fact that when urination became painful, difficult or impossible, the male being was driven to that desperate necessity which is the mother of invention.

Thus, among the ancient Hindus, probably over a thousand years before Christ, we find the catheter used to relieve retention of urine; the sound to dilate strictures of the urethra, and the operation of perineal lithotomy fully developed and widely practised.

Sucruta's manuscript giving us details of the operation of lithotomy was written about B. C. 600. It is interesting to note that the perineal operation for stone, which is so skilfully performed by the native lithotomists of India to-day, is practically the same as that described by Sucruta 2500 years ago.

In the writings of Hippocrates (B. C. 460) we find accurate descriptions of the formation of urinary calculi, of lithotomy and of nephrotomy; and this at a time when practically no other operations upon abdominal viscera were attempted. The history of major surgery may, therefore, be said to have its beginnings in the work of the early urologists, and to owe its inspiration to them.

By the Hippocratic oath the general practitioner of medicine was required to promise never to perform lithotomy, but to leave the operation to those who made a specialty of it—the so-called lithotomists.[2]

Celsus shows by his writings at the beginning of the Christian era that operations for calculus, retention of urine and urinary fistula, were well developed; and Galen (B. C. 130-210) and Aurelianus (B. C. 300) describe certain progress in urology; but during the long years of the middle ages "while medicine was in the hands of the monks who disdained surgical operations," the "Lithotomists" alone kept up the surgical spark. During this time certain inhabitants of the town of Norcia developed such skill in lithotomy that they were called to all parts of Europe, and were frequently official employees of municipalities where they attained great fame, thus being the first public health officers.

The operation for stone, which had thus far been entirely in the hands of these itinerant laymen, was finally adopted by the medical profession through the work of Germain Colot about A. D. 1460.[3] Colot was a French surgeon of renown. Declaring it a disgrace that an operation of such importance should have been so entirely neglected by surgeons, he went to Italy, ingratiated himself with a family of lithotomists, learned their methods, and then returning to France, instituted a series

[1] Oration on Surgery. Southern Medical Association, Ninth Annual Meeting, Dallas, Texas, November 8-11, 1915.

[2] See Guitéras: The Evolution of Urology, N. Y. Med. Jr., Oct. 11, 1902, which has been freely quoted.

[3] Fisher: Annals of the Anatom. Surg. Soc., 1880.

of experiments upon the cadaver, the results of which he reported to the Medical Faculty of Paris, and at Court. He then applied to King Louis XI, who granted him permission to operate on a criminal condemned to be hanged.

It is interesting to note that this historic operation, which was carried out in the presence of the King, was performed in the churchyard. The patient showed great fortitude, was well in two weeks, and saved his own life, besides saving the lives of future generations by securing this operation to surgery.

Colot was decorated by the King, appointed the Royal Lithotomist, and became the father of a family who furnished France with its chief lithotomists for three hundred years. Although medical men, they kept the secret to themselves just as the Norcians had done, and were consequently reviled by the rest of the profession until François Colot, the last of the family, gave the secret to the medical world in 1727.

The general surgeons, however, seemed unable to make a success of lithotomy, which was still the gravest and most difficult operation in surgery, so that again non-medical operators flourished—the chief of whom was a monk named Frère Jacques who, marvelous to relate, invented and introduced the lateral operation for stone, which requires far more precision and expert knowledge of anatomy than the median operations previously used. The story of Frère Jacques, his meteoric rise at court, the crowds that assembled to see his lightning-like procedures, his admission to the great hospitals of Paris over the protest of the surgeons, and his final departure in disgrace because most of his patients died, is one of the most thrilling histories of surgical literature.

Although discredited in Paris he evidently flourished elsewhere, as he is said to have cut more than five thousand for stone, and often to have operated upon sixteen patients in one afternoon. He died in 1720, and his operation, perfected by the great Englishman Cheselden, is notable as being based on the finest anatomical principles, in that it avoids injury to such important structures as the external sphincter, the cavernous tissues of the bulb, and the crura of the penis, thus preventing incontinence and sexual impairment.

To this day the non-professional lithotomist exists in the East; and even in Scotland, only a century ago, Mr. Adams stated that "there was an old miller who was very famous for cutting persons for the stone." For some inexplicable reason the major things in surgery were largely left for uneducated operators—even Ambroise Paré was a blacksmith. Paré learned lithotomy from a Colot, and was one of the most accomplished in its performance.

My prolixity on this subject is due to my desire to point out surgery's debt to the ancient and mediæval urological specialists who, almost alone, kept going the flame of major surgery when the forces of religious and medical superstition would have extinguished it. Indeed it was not until John Hunter came that the solid foundations of scientific surgery were laid. It is interesting to note that at that time there were only two great surgeons in England, William Cheselden and James Douglas, both of whom owed their reputation chiefly to their operations for stone. Hunter became the pupil of one of these, Cheselden, the originator of the present "lateral operation for stone," which he was able to perform in 54 seconds.

It is probable that this association with Cheselden, the foremost urologist of his day, inspired Hunter to undertake his masterly researches on genito-urinary diseases, and to collect at such great pains the wonderful specimens of urinary disease, particularly of calculus, enlarged prostate and kidney, which form the most striking and valuable part of the pathological work left by him. It is very evident, from a study of the clinical works of Hunter, that his private practice was largely genito-urinary in character. It was Hunter's practice to admit young men into his home for a long period of years as private students of surgery, and it is not surprising to find that of these men who afterward became famous, and caused English surgery rapidly to eclipse the rest of the world, the majority were particularly interested in genito-urinary surgery. I need only refer to John Abernethey's celebrated treatise on syphilis, Sir Astley Cooper's classic on the testicle, Sir Everard Home's two volumes on the hypertrophied prostate gland, and James Wilson's anatomical researches on the external urethral sphincter.

The history of urology in France during the eighteenth and nineteenth centuries, as in England, contains the names of almost all the celebrated surgeons, for here again it was the most difficult and dangerous surgery performed. Commencing with Guy de Chauliac, we find Colot, Paré, Dionis, Le Dran, Desault, Dupuytren, Nélaton, Chopart, Civiale, Leroy, Heurteloup, Mercier, Maisonneuve, Guyon, Pousson and Albarran. The most important invention of this time was that of stone crushing by Civiale, in 1818. This subject was further developed by Leroy, Heurteloup in France, and by Thompson and Weiss in England. Mercier's work on obstruction at the vesical neck, and the ingenious instruments he devised, were of great importance; but to Civiale first, and later to Guyon, the medical profession owes most of the solid foundation of urology upon which so wonderful a superstructure has lately been reared.

Sir Henry Thompson, in England, a brilliant pupil of Civiale, pursuing the careful research methods of John Hunter in anatomy and pathology, did more perhaps than any one man in the nineteenth century to bring urology into the very forefront of surgery, and to inspire men to devote their lives to this special branch.

While the school of Thompson in England, and that of Guyon in France, were increasing in vigor and force in the latter part of the last century, America and Germany had awakened and were fast becoming powerful factors in the rapid advance.

Early in the past century America developed a remarkable coterie of lithotomists. The greatest of these was Dudley of Lexington, Kentucky, who had the remarkable record of 207 cases with only 6 deaths—1 in 34½, or less than 3%—Valentine Mott, the greatest New York surgeon, had 7 deaths in 162 cases, and Nathan R. Smith of Baltimore 3 deaths in 45 cases. In a recent volume, "Kentucky Pioneer Lithotomists," Dr. A. H. Barkley tells of the wonderful work done by McDowell,

Dudley and Bush. McDowell learned lithotomy from John Bell in Edinburgh, in 1794, and had 28 cases without a death. Dudley, who followed him, was a student of Sir Astley Cooper in London, in 1813, and had only 3 deaths in 225 lithotomy cases, according to Barkley, and one series of 100 cases without a death—a record which has never been equaled. Bush worked with Civiale in Paris, in 1839, and became America's foremost litholopaxist. These three surgeons, all residents of Lexington, Kentucky, and occupying the same office, formed one of the most remarkable medical groups in America. The total for the United States, as collected by Gross in 1850, was 851 cases with only 1 death in 20; whereas, according to Coulson, in Europe the proportion of fatalities was 1 in 5.

The greatest of all American surgeons, Samuel D. Gross, was himself an eminent lithotomist, and his principal work, "Diseases of the Bladder, Prostate and Urethra," published in 1850, still ranks as a classic. Although a professor of general surgery and a prolific writer on many subjects, his most brilliant work was in urology; and he may really be said to be the father of American urology, though Van Buren and Gouley, and later Otis and Keyes, of New York, and Bigelow, of Boston, exerted a more continuous influence. These men were the peers of the European urologists, and by their original contributions soon placed America in the forefront. Bigelow's classic researches on lithotomy challenged the admiration of the world and revolutionized the treatment of vesical calculus. Each of these men surrounded himself with ardent young fellows who have since formed the New York, Boston and Philadelphia "schools" of urology.

Austro-Germany lay dormant during this period of great activity in England and France, until the latter part of the last century, when Simon at Heidelberg began his epoch-making work on the kidneys. A young woman with a ureteral fistula appealed to him for relief. The only possibility of cure lay in removing the kidney; but no one knew whether life with only one kidney were possible. Could the repair of the renal artery ligated so close to the great aorta stand the pressure after absorption of the ligature? Might not emboli or peritonitis bring on early death? No one knew. In order to find out, Simon nephrectomized 30 dogs, and thus not only put the operation on a sound basis, but led the way to animal experimentation which has revolutionized surgery in recent years (another debt of surgery to urology). The operation was then carried out on the patient with perfect success in July, 1869. The great Bruns carried out the same procedure two years later with, however, a fatal result, and the next patient of Simon himself lost her life. The tragic story of this case is so beautifully told by Pilcher[4] that I cannot refrain from giving it again.

The patient was a brave little woman of Savannah, Georgia, who was sent across the Atlantic to Simon to be cured of a calculous pyonephrosis. "All previous authority had condemned such a procedure; never before had it been attempted, not until the operation was happily finished did the apprehension leave him that he (Simon) might yet find himself confronted by the conditions which his predecessors had described as unsurmountable. For three weeks the convalescence proceeded without disturbance, and the wound had contracted until it was now insignificant. Simon, however, shared in that spirit of the older surgeons, which prompted them to examine the cavity with their own fingers. They called the practice training their *tactus eruditus*. No unfortunate patient could have a cavity opened or a wound made without the bystanding surgeons being invited to put their fingers into it—the omission was a breach of professional courtesy. The boldness and enterprise of this Heidelberg surgeon had carried him further than most surgeons. He had passed his hand and forearm through the anal sphincter, and along the large intestine until he could feel the kidneys and liver of his patient; he had made a practice of dilating the female urethra with his forefinger which he carried into the bladder and by it guided a catheter into many a ureter (thus becoming the first to do ureteral catheterization). Where his fingers had been previous to his approach to the bed of his now fully convalescent nephrectomized patient, on this her twenty-first morning after the operation, who can say? The vexed question of antiseptics, rubber gloves, and surgical cleanliness had not yet begun to trouble the surgical mind. So much the worse for this little woman from Georgia, and her dreams of future health. As the dressings were being changed, the temptation to explore was too great, and in went his finger. Within a few hours came a chill and fever. For 10 days the battle between the body cells and the invading micro-organisms continued, and then death!"

Had Simon only heeded Lister, who four years before had published his great work on antisepsis (1867), the result might have been different. A similar fatal case of nephrectomy for calculus, in the hands of the great Von Bruns, put an end to kidney surgery for some time, and as late as 1879 Pilcher was able to find only 15 cases in the literature. But Lister's teachings had at last found an audience, and renal surgery was among the first of the great fields of major surgery to feel its influence. In 1885 Samuel D. Gross, in one of the most masterly clinical analyses ever made, collected 450 different operations on the kidney, and placed the whole subject on an enduring basis.

The next great advance in urology came from Austria—I refer to the invention of the cystoscope by Nitze in 1876. This instrument, which could not be perfected until Edison in 1879 brought forth the incandescent electric bulb, has not only revolutionized urology and brought it from darkness into light, but also has done more than any one invention to stimulate exactness in diagnosis, and the use of electricity in medicine.

Truly marvelous are the instruments which followed in steady succession. The ureteral catheterizing cystoscope, the operating cystoscope, the photographic cystoscope, the stone-crushing cystoscope, the fulgurating cystoscope, the rongeur cystoscope, the cauterizing cystoscope, the urethroscope, the bar-exercising endoscope, the laryngoscope, the otoscope, the gastroscope, the proctoscope, and electric ophthalmoscope, need

[4] Annals of Surgery, 1900, vol. 31.

only to be mentioned to indicate the tremendous activities which have been evoked by the development of the first cystoscope. In this great advance the geniuses of Germany and America have shared honors about equally. The great desideratum, catheterization of the ureters in the male, which had been looked forward to longingly but vainly for generations, was finally accomplished by an American, my predecessor at The Johns Hopkins Hospital, Dr. James Brown; and another Baltimorean, Howard A. Kelly, made ureteral catheterization in women possible and easy, and led to the brilliantly rapid evolution of renal and ureteral surgery that immediately followed. Time will not permit me to do justice to other American confreres who have done so much for this subject—Tilden, Brown, Otis, Belfield, Koch, Valentine, Lewis, Buerger, McCarthy, Squier and others.

It is important, however, that I refer to the great diagnostic methods that have been evolved as a result of the ureteral-catheterizing cystoscope. The mere separation of the secretions of the two kidneys was a great step in advance, enabling one to recognize the diseased kidney and to discover early tuberculosis, unilateral hematuria and, with the development of the X-ray and pyelography, various hitherto unrecognizable lesions. But it did not offer sufficient evidence concerning the other kidney. It did not remove that old dread that the remaining kidney might be unequal to the task of sustaining life when its fellow had been removed. For almost 20 years surgeons have been searching for an ideal functional test of the kidneys, and various drugs, generally dye-stuffs, have been brought forward. Casper and Richter, who introduced the phloridzin test, deserve great credit for impressing upon the profession the need of functional tests, although their phloridzin test proved unsatisfactory. The ideal test seems now to have been discovered; I refer to the phenolsulphone-phthalein test, the result of the work of four of my *confrères* on The Johns Hopkins Faculty—Professor Remsen, who discovered the drug, Professor Abel, who showed that it was almost entirely eliminated by the kidneys, and Dr. Rowntree and Dr. Geraghty, who proved its clinical value by a long series of experimental and clinical researches.

Thanks to this splendid example of co-operative and consecutive effort between various departments, we now have an accurate method of determining factors of safety in renal surgery, and are in a position to know beforehand what should be done, and whether it is safe to do it.

A glance at the mortality figures during the past 30 years will show the transformation which the cystoscope, the ureteral catheter and the functional test have accomplished in kidney surgery. Gross, in his collection of 450 cases, found the mortality for nephrectomy for various diseases to be between 20% and 60%. In America now it is between 1% and 5%.

The Prostate.—The surgery of the prostate was even later in its development than that of the kidney. Although Covillard in 1638 removed an hypertrophied lobe after perineal lithotomy, Sir Henry Thompson, whose researches on the anatomy and pathology of the prostate are still classics of great value, stated as late as 1887 that successful prostatectomy was unknown.

It is almost tragic to hear this great urologist say: "I desire extremely to see such a result; I have on four occasions removed considerable portions of the prostate, but without success. I have traveled considerable distances abroad expressly seeking it, but without success." But at the time that he was writing younger forces were at work, and as a result of the cumulative efforts of Belfield (1887), McGill (1889), Goodfellow (1891) and Fuller (1894), prostatectomy became an established operation.

More accurate methods, based on anatomical landmarks and pathological studies, have greatly improved the perineal operation, so that the mortality rate which was 15% 20 years ago, has fallen to about 3% for conservative perineal prostatectomy in our clinic. In a recent study of 775 cases, I found 30 deaths, or 3.8%. Only eight deaths occurred within the first week after the operation, and 10 after a month. These figures show how benign prostatectomy can be made.

The careful study of cases, particularly as regards the kidneys, has had even more to do with the lowering of the death rate than a carefully planned anatomical operation. An early recognition that a large residual urine was a danger signal, especially if the patient had never been catheterized systematically, enabled us to so reduce the operative risk that it was possible to operate upon 128 consecutive cases without a death. Now that we have shown that the phthalein test gives an accurate index to the state of the kidneys, a means of determining when the operation may be safely performed is at hand, and with careful preparatory catheter treatment makes it possible eventually to operate on almost every case. One of our recent patients was 93 years old.

One of the most important urologic problems of the day is cancer of the prostate, which is now known to occur one-fourth as often as hypertrophy. It shows itself in early cases as a localized, very hard area in the posterior portion of the prostate, and if routine rectal examinations were made many early cases of cancer of the prostate would be detected. Ten years ago it was supposed to be beyond surgical relief. Now we have a well-established radical procedure in which, although the whole of the prostate, the seminal vesicles and the neck of the bladder are excised, the anastomosis between bladder and stump of membranous urethra can be so effectively made that neither stricture nor incontinence follow.

We have now apparent cures in 10 cases of cancer of the prostate—most of them over five years', and one of 10 years' duration—the percentage of cures being about 65. I predict a great future in cancer of the prostate.

Time does not permit to mention more than a few of the conquests of urology, but I must say a word in regard to the epoch-making advances that have been made in the treatment of vesical tumors. Even five years ago all statistics of results after operative excision of bladder tumors, both benign and cancerous, were so bad that Watson was boldly proposing cystectomy—complete removal of the bladder for almost all bladder tumors. The remarkable results obtained by Nitze with his cystoscopic cautery snare had been lost sight of. Then came Beer's report on the use of the high frequency electric spark, which causes most papillomatous tumors to disappear as

if by magic, and the statistics of results were transformed. When one sees a bladder half filled with sessile papillomata, each the size of a small walnut, become normal with not even a scar to show the site, one realizes that a wonderful therapeutic agent has been discovered.

Radium, too, is a force of marvelous power, the limits of which are little known, but which occasionally works seeming miracles. We have been using radium by means of special radium-carrying operative cystoscopes, in conjunction with the high frequency treatment of vesical tumors, and prostatic carcinomata, with really promising results.

The surgery of the seminal vesicles has also made rapid strides. Fuller deserves much credit for the persistency by which he has finally impressed upon the medical profession the important rôle of chronic infections of the seminal vesicles and prostate in the etiology of rheumatism, arthritis and heart disease. His operation of drainage of the seminal vesicles has accomplished wonders in curing hitherto intractable "rheumatic" infections. Recent improvements of the perineal prostatectomy technique have been applied to vesiculotomy and vesiculectomy, and made them accurate procedures under visual direction, removing all guesswork. I have extended this operation recently to the complete excision of tuberculosis of the seminal tract with the result that it is now possible to remove entirely extensive tracts of tuberculous infection, which were previously beyond surgical relief, and with the same splendid results which have followed nephrectomy for tuberculosis. It is no longer necessary to relegate these sufferers with seminal tuberculosis to a life of slow torture and ultimate death, for many will get well after excision of the entire infected genital tract, just as nephrectomy with ureterectomy has been shown to cure tuberculosis of the urinary tract.

Litholapaxy, which was the great contribution of America over a quarter of a century ago, has been simplified and facilitated by the invention of the evacuating lithotrite of Chismore, and the addition of the cystoscope has now made it possible to crush, evacuate, cystoscope and pick up a remaining fragment, all with one introduction of the instrument. The use of the median-bar excisor, or "punch," makes the cure permanent by removing the small bar or contracted vesical orifice, which in the past has been such a fertile cause of calculus recurrence.

I have already referred to the marvelous array of cystoscopic instruments which have been invented. It is now possible with the catheterizing cystoscope not only to collect the separated kidney urines, but to dilate strictures in the ureter, to facilitate the passage of calculi or even fracture them with electrical ureteral sounds, and to fill the ureters and kidney pelves with thorium nitrate, and thus obtain x-ray photographic demonstration of various lesions which were formerly hidden. The operative cystoscopes make possible the removal of cysts, tumors, specimens, foreign bodies and prostatic lobules, the division of strictured ureteral orifices, and the removal of small calculi from the ureteral orifices and from diverticula. Urology possesses unquestionably the most perfect diagnostic instruments and laboratory methods in the entire field of medicine and surgery. But what has brought about this splendid achievement in accurate diagnosis and skilful operative relief? First, the fact that high class surgical work requires high class mechanical and technical men, and, if possible, personal training and skill in such by the operators themselves. With the mechanical talent, ability as draughtsmen to convey one's conceptions and ideals goes hand in hand, and a survey of the literature shows many remarkable instances in which mechanics or art, or both, were developed to a marked degree in the great men of medical science. I need mention only a few: Vesalius, the father of anatomy; Ambroise Paré, the father of surgery; Karcher, the inventor of the microscope; Cheselden, the great English surgeon, who designed Putney Bridge; Scarpa, Henle, His, Charles and John Bell, Leidy and Lister, all of whom illustrated their own works with beautiful drawings; Charcot, the great neurologist, and Seymour Hayden, one of the greatest painter etchers of all time, as well as a great surgeon.

Urology, probably more than any other specialty, owes much to mechanics and art in her devotees. The four moderns who have done most to develop the science are Nitze ("der grosse Techniker"), Henry Thompson, Henry J. Bigelow and Felix Guyon. All of these epoch-making men were great mechanics, inventive geniuses and idealists, whose spirits were never satisfied with the existing order, but constantly strove for perfection of detail, as well as broad advancement in the field.

The second factor in the modern advancement in urology has come about as a result of a closer co-operation between clinic and laboratory; for there is no branch of surgery which offers greater field and opportunity for varied laboratory work than urology.

A recognition of this fact led us, when preparing the plans for the James Buchanan Brady Urological Institute in Baltimore, to provide a most varied assortment of laboratory and research facilities presided over by well-trained whole-time men. Not only were laboratories for pathology, bacteriology and clinical microscopy provided, but also for chemistry, physics and animal experimentation, with a well-equipped machine shop to make new apparatus and surgical instruments. The men presiding over these scientific activities have been called from various universities; and, an internist, to work on borderline subjects between medicine and urology in both a clinical and experimental way, also devotes his entire time to the institute.

This close co-operation between scientific and clinical staffs has already proved of inestimable value, and the free discussion of the problems has been of much mutual help and inspiration.

All surgery has become so perfect in technical detail that advancement in the future will come from researches in the broader fields of chemistry, physiology and experimental medicine and surgery, but these can be very fruitful only when closely associated with the clinical work, and when the clinicians themselves become laboratory men and experimentalists. Such it has been our effort to provide for, with the belief that urology furnishes a most fertile field for research, and in the full expectation that it has a most brilliant future ahead of it.

WHAT WAS THE ENGLISH SWEATING SICKNESS OR SUDOR ANGLICUS OF THE FIFTEENTH AND SIXTEENTH CENTURIES?[1]

By Arpad G. Gerster, New York City.

Member of the Charaka Club.

Contemporary writers differ about the exact date of the landing at Milford Haven of the Duke of Richmond in the year 1485. Presumably the date of August the first is correct, as this is the day mentioned in the Historia Croydenensis.[2] The invading army proceeded by way of Cardigan towards Shrewsbury, where it crossed the Severn; thence to Newport, Stafford and Lichfield, where it was encamped by August 18 on swampy, fenny land. Bosworth field is not far from Lichfield, and here was fought the battle which ended in the defeat and death of Richard III. Three weeks had elapsed between the landing and the battle, and on the march about 200 miles had been covered by Richmond's men. In view of the slow movements of the military forces of that day, caused by the weight of armaments, impedimenta and bad roads, the march was a very rapid one, involving more than usual hardships. During the passage from Havre to Milford Haven, which took seven days, much sickness developed among Richmond's mercenaries, and after the landing a large but unknown number of them had to be left behind along the line of march. Many of these died shortly after having fallen ill, and apparently the malady was rapidly transmitted to the inhabitants, spreading terror in all directions, for we know that Lord Stanley, temporizing, and peremptorily summoned by Richard, pleaded as an excuse the ravages of the new distemper. After his victory, King Henry VII in four days proceeded to London; and probably, according to the custom of the times, his army was disbanded. By September 21 Bacon[3] reports a general prevalence of the deadly malady in London, where during five weeks "infinite persons," and according to Stow[4] "a wonderful number," succumbed. By the end of the year the epidemic had spread with undiminished virulence all over England, sparing neither poor nor rich. Many were its victims among the clergy, gentry and nobility. When it appeared at Oxford, teachers and students fled. No exact data exist regarding the number of the dead, but it must have been very great. The medical profession was overwhelmed by the new disease and stood helpless in the face of the calamity.

Baker[5] states that "many thousands died," and "no physick afforded any cure." The contemporary authors are silent about the part played by the medical men in this epidemic. Even the famous Linacre, founder of the College of Physicians, who in his young days had witnessed the panic caused at Oxford at the first invasion of the disease, and who lived through two more of the epidemics, makes in his writings no mention whatever of it. This peculiar circumstance might be explained by the fact that the men who revised the study of the ancient medical writers—Linacre one among many—devoted more time and effort to diacritical research in the writings of Hippocrates and Galen than to the actual observation of maladies.[6] The most learned ones among them were rather profound linguists than adroit physicians. Erasmus, a close and admiring friend of Linacre, did not fail to perceive this anomaly; nor did he hesitate to launch a shaft of sarcasm at the linguistic pedantry of the learned of his day. The famous passage on page 200 of his "Laus stultitiae" is generally accepted as applicable only to Linacre. "I have known a certain man of universal learning (polytechnotaton) of Greek, Latin, Mathematics, Philosophy, Medicine, and all the regal pomp of erudition, although past 60, to neglect everything during more than 20 years, so that he may freely torture and crucify his mind at digging in grammars; who has directly stated that he would consider himself fortunate if permitted to live long enough to be able to explain, without any doubt, in what manner the eight parts of human speech could be certainly distinguished, one from the other."

Whether with or without the aid of the profession, the common sense of the laity finally evolved a method of treatment which at least proved to be harmless and was generally accepted all over the kingdom. Its essence was the avoidance of all violent methods and remedies, abstention from food, scanty potations of mild beverages, and confinement in bed during 24 hours under light coverings. The fear of suppressing the perspiration suggested the rule that those attacked in the daytime should seek their beds with their clothes on; those that sickened during the night were not to rise; all avoiding exposure to the outer air of any part of the body except the face. By New Year's Day, 1586, the disease had vanished from England.

Before proceeding further it will be necessary to add some facts collected by the indefatigable Hecker that may have had an important bearing on the development of this pestilence. The first one is this: That since the year 1482 there had existed in France a murderous disease which is called by Mézeray[7] "une

[1] Dr. I. F. C. Hecker: Der Englische Schweiss, Berlin, 1834.

[2] Joannes Tell: Rerum Anglicarum Scriptores veteres, Oxon., 1684.

[3] Francis, Lord Verulam: The Historia of the Raigne of King Henry the Seventh.

[4] John Stow: The Annales of England, etc., London, 1592.

[5] Sir Richard Baker: A chronicle of the Kings of England, etc., London, 1665.

[6] The later Greek authors, according to Hecker, ignored the existence of small-pox during four centuries, because there was no mention of the disease in the writings of Galen. Linacre might have found an excellent description of the sweating sickness in Aurelian, a Roman physician of the fifth century, whose language, however, characterized by many romance elements, was considered corrupt; hence the author was ignored.

[7] François Eudes de Mézeray: Histoire de France, 3 tomes, Paris, 1682, t. 2, p. 746.

dangereuse et mortelle maladie," which, "though not contagious, attacked indifferently great and small." It was a species of hot and frenetic fever, which suddenly inflamed the brain and scorched it with such cruel pains that some broke their heads by butting the walls; others jumped into wells, etc. The chroniclers all report that the year 1485 was preceded all over Europe by five excessively wet years with continuous rains and many inundations; the crops failed to mature in most places, and famine stalked through all Europe. The year 1486 was the sixth wet year in succession, and inundations of the Tiber, the Po, the Danube and the Rhine were recorded.

The composition of Richmond's army also deserves attention. Its bulk consisted of armed mercenaries originally licensed by Louis XI, free lances—that is, the scum of humanity—to join whom there flocked all that were given to crime and vagrancy. Mézeray says of them: "There were in Normandy alone some troops of free lances (francs-archers) belonging to those permitted by Louis XI, and who travelled the country; and being joined by many vagrants they ravaged all the land, so that the fear arose that the evil might be spread to adjoining provinces; but just then a good occasion presented itself to deliver France of these robbers, and he (Charles VII) gave him (Richmond) all these free lances and brigands of Normandy to the number of 3000."

It is easy to imagine what the hygienic conditions must have been in the midst of which lived this armed mob. The crowding and uncleanliness on board the small vessels during the week elapsing between July 25 and August 1 must have been excessive, and must have served as hotbeds for the spreading of the disease.

Let us cast a glance now at the manner of life that obtained in the England of those days. In great contrast with present conditions, the state of untidiness of English dwellings was truly Scythian. Erasmus, who never was contradicted regarding the matter, writes: "The flooring of the houses is usually beaten clay; this is spread with rushes, which are rarely removed, additional layers being added to those already fouled, which sometimes remain undisturbed for the period of 20 years; this mixed with fishbones, vomit, and other filth, becomes saturated with human and canine urine." Undoubtedly there is in this description much exaggeration, but the universal use of rushes on the flooring of the houses of even the wealthy is a well attested fact. The prevalence of vermin among the poorer sort, as to-day in the near East, was an accepted condition, against which even the better situated struggled with uncertain success. As we know the magnitude and difficulty of the task of keeping present-day armies free from vermin, we shall have no difficulty in imagining conditions in 1485. To recapitulate, these factors have to be kept in mind: The importation of a febrile disease from France by an undisciplined army of freebooters, who had spent a week at sea closely confined and crowded into the unclean, small vessels of the period; the entrance of this infected army into England, exhausted by the murderous wars of the Roses, and impoverished and starved in consequence of a series of failures of crops, which had never matured during years of rainy and cold weather; the dwellings, almost exclusively built of wood, or wood and bricks, small, crowded, filthy, and all more or less infested with lice, ticks and bedbugs. The prevailing cold weather was, of course, a powerful incentive to crowding and uncleanliness. Here we have a combination of factors unsurpassed to serve as the hotbed for an epidemic.

Of the writers of the period who recorded the events of the malady, it might be said that all of them were laymen. From Linacre we have not a line written on the new disease. The descriptions of the symptoms of the disease and of the manner of its spreading are very scanty and unsatisfactory. They are also often contradictory; but this much can be gathered from them: That the onset was sudden; that the fever was excessive, initiated by a chill, accompanied by headache, anorexia, vomiting, backache and pain in the muscles and long bones; that it attacked all ages and conditions; killed in the first access of pyrexia not only those enfeebled by want or exposure but many of the better situated and seemingly strong, who indulged in perpetual gorging of almost exclusively animal and highly-spiced food, together with alcoholic excesses, the chief amusements of polite society. The consumption of beer by the populace was not diminished but rather stimulated by the general misery, and the habitual beverage of the well-to-do consisted of strong wines imported from Greece, such as Muscatel and Malmsey. Vegetables were not cultivated, so that Queen Catherine had to import soup-greens and lettuce from the continent.

At the acme of the fever coma supervened, which was soon followed by death, sometimes within two hours after the onset. All the authors lay stress on the circumstance that after the lapse of exactly 24 hours an excessively profuse and nauseatingly foul sweat broke out all over the body of the patient, after which there followed either a remarkably speedy recovery or a slow and protracted convalescence. A return of the fever was very frequent, and occurred once, twice or oftener, as many as 12 recurrences being mentioned. Few of the writers, however, lay stress on the phenomenon of recurrence, their attention being entirely dominated by the more obvious symptoms—the rheumatic pain, the headache, the burning fever, the coma and the ill-smelling profuse sweat. Most of them, Bacon among the rest, did not consider the disease epidemic (meant for contagious) like the plague, because many were attacked who had had no contact whatever with the sick. All writers accepted the theory that the malady was due to an atmospheric poison, precipitated from the perpetual pall of fog and rain. Remarkable is the unanimity of all the contemporary authors regarding the sudden and complete cessation of the disease.

The second invasion by the sweating sickness occurred in 1506, only seven years after the terrible destruction of life wrought in 1499 by the plague. In London alone there died 30,000 persons, and the King fled the country, retiring to Calais. On the whole, this epidemic was milder than the first.

The third epidemic appearance of the sweating sickness took place in the year 1517. It ravaged the country during six months, having spread to the frontiers of Scotland. Among the general conditions that might have favored its development,

the following may be mentioned: The end of the reign of Henry VII and the beginning of that of Henry VIII witnessed a remarkable development of England's industrial life. The conversion of much arable land into pasture caused a noteworthy migration of the farming population to towns and cities. Large numbers of Genoese, Lombard, French, German and Dutch tradesmen had settled in England. Under the stimulus of their skill, enterprise and industry, a flourishing trade established itself. Great prosperity but excessive crowding resulted. As the invasion of the towns by the starving country people could not be checked, much unemployment, poverty and squalor followed. All the towns were noted for their narrow and uncleanly streets, and a large proportion of the poorer inhabitants were housed in damp and incommodious cellars. Contrary to the conditions of 1485 and 1505 the weather was not unusually rainy, and the crops were satisfactory. The opulent manner of eating and drinking of the industrial classes was stimulated by prosperity, a wellbeing from which the unemployed and vagrant poor were excluded—a condition essentially unchanged to this day. Bloody riots of the unemployed in London occurred in April and May, 1517, remembered under the name of the "Insurrection of evil May day." In July the sweating sickness broke out among the poor, and Goodwyn[8] says, "Of the common sort, they were numberless that perished by it." The wealthy, however, did not remain exempt. Ammonius, the King's secretary, having boasted to Thomas More of his immunity, due to simple habits, died two hours afterward. Of the court, there died Lords Grey and Clinton, besides a large number of attendant nobles and other persons of the household. The King fled to the country, frequently changing his abode, whereto he was followed by lugubrious news, reporting that in this town or that one-third or one-half of the population had died. The usual festivities of Michaelmas, even those of Christmas, were abandoned. Thomas More's family was not spared, his daughter, Margaret Roper, having fallen sick. She recovered, however. In one of his letters to Erasmus, her father's distress found expression in this sentence: "Almost all being stricken within a few days, most of my best and honored friends were taken away." The acme of the distemper was reached six weeks after its beginning. Oxford and Cambridge suffered great loss in the deaths of their best teachers. The Scotch and Irish were spared, but the malady extended to Calais, where only the English were affected, the French remaining untouched.

In 1528 the sweating sickness appeared for the fourth time in England. This epidemic became noteworthy for several reasons. First, it was more destructive than the preceding ones; secondly, it spread to Holland, Northern Germany and the Scandinavian countries. As to the meteorological conditions preceding and during the disease, all writers of the time agree that 1527 and 1528 were excessively wet and cold years, not only in England but over most of Northern Europe, so that Count Newenar, Canon of the See of Cologne, wrote as follows: "There were in that and during the preceding years so many storms, inundations, cold spells combined with perpetual humidity, that it seemed as though the climate of Great Britain had taken possession of Germany."[9] In England there was incessant rain during November and December, 1527, and in January, 1528, resulting in extensive inundations which killed the winter crops. The spring sowing was scarcely finished when, in April and May, it rained continuously for eight weeks. Thick fogs covered the country in the pauses between the rains, and destroyed the last hope for a harvest. Such weather prevailed during a succession of years over most of Europe. The chronicler of the city of Erfurt reports that after an unusually mild winter spring set in so early that on St. Matthew's day (February 24) everybody was wearing posies of wild violets. The rest of the spring and the summer brought an unending succession of rainstorms, causing widespread inundations and a total failure of the crops. The resulting famine was appalling, cattle and even the birds of the air dying in great numbers. Among the expedients resorted to during the calamity may be mentioned the following: When the season drew to its end parts of the green crops that had not rotted out were cut, and the ears of grain were parched in ovens to be eaten in the shape of porridge.

The "Great Dying" began in London during the last week of May, 1528, spreading thence all over the realm. The virulence of the malady was terrifying, most of the afflicted passing from wellbeing to death within five or six hours. Quarter sessions were suspended, and notwithstanding general protestation, St. John's day solemnities and feasting were suppressed. From among the courtiers, Sir William Compton and William Carew, both chamberlains to the King, died in quick succession; and when Sir Francis Poynes, the recently returned ambassador at the Court of Spain, succumbed, the King fled London and wandered about the country until he settled down in the solitudes of Tytynhangar, surrounded by a chain of bonfires, kept up day and night. The combination of famine, of a depressing humidity, and the consequent crowding together of starving multitudes in filthy habitations, were the leading factors of this epidemic. There exists no reliable record of the mortality, but it must have been greater than in any of the preceding epidemics, as some of the writers use the term, "universal depopulation." The epidemic itself was dubbed by the people "The Great Dying." It is a strange fact that not one of the writers of the time noted the duration of this epidemic, and it is fair to assume that sporadic croppings up of minor foci and of scattered single cases dragged out the disease into the year 1529, so that the precise ending could not be determined.

In this year, with the arrival of the skipper Hermann Evers from London on July 25, the disease made its first appearance in Hamburg. Twelve of the ship's company died, some at sea, four after their arrival in Hamburg. The epidemic lasted 22 days and caused the death of 1100 of the inhabitants. The count is exact, because this was the attested number of coffins delivered by the joiners' guild. The distemper appeared in

[8] Francis Goodwyn (Bishop of Hereford): Annals of England, etc., London, 1675, p. 23.

[9] Hecker: Foot-note, p. 93.

quick succession at Lübeck, Zwickau, Stettin, Danzig, Augsburg, Strassburg, Köln, Frankfurt am M., Marburg, Göttingen and Hannover. The cropping out of the disease as to choice of locality was capricious, but this much is evident, that the further south it extended the milder and rarer it became. Amsterdam was stricken on September 27. From there the mischief spread over all of Holland and Belgium. Alkmaar and Waterland alone remained intact. At about the same time the sickness invaded Denmark where, in Copenhagen, on one day, September 29, 400 persons died. It also appeared in Norway and Sweden. Within three months of its first appearance in Scandinavia the epidemic ceased. The strange silence of contemporary English medical writers about the sweating sickness, which they had abundant opportunities to observe during the preceding epidemics, has been mentioned before. The learned doctors of the universities were under the scholastic spell of the ancients. Whatever did not occur in Hippocrates or Galen did not interest them; it was simply ignored. The number of these highly learned linguistic doctors was small, and not all of them practised. The great bulk of medical work was in the hands of licensed or unlicensed, mostly illiterate, ignorant surgeons and barbers. Medical writing was in Latin and inaccessible to the commonalty. The Catholic faith was still unshaken in England; nay, the King had entered the lists against Luther's heresy, thereby earning the title "Defensor Fidei," still attached to the English crown.

By the time the disease penetrated to Germany, however, the country had been in the throes of the great reformation full 12 years. Religious controversy of the most passionate character was in full blast, its main vehicle being innumerable sheets and broadsides printed in every town of note. Gradually other than religious questions of public interest were seized upon by the pamphleteers, who used this convenient medium to spread their opinions on politics, trade, education, national economy and what not, among an eager and awakened public. The new and murderous disease became naturally a subject of much controversy and of universal interest. Though most of them have disappeared, there is still extant a rich array of pamphlets and broadsides printed in Holland and Germany from the pens of medical men, humble and eminent, and also from a host of laymen of various character. There were even official publications, such as that of the great Council of Berne, whose purpose was to combat the universal terror and to give therapeutic directions of a mild and prudent nature. From these pamphlets we gain many valuable observations regarding the facts of the malady and of the curative measures employed against it.

As the valuable experiences gathered in England were unknown, the appearance of the epidemic found both profession and people of the Continent unprepared. The first pamphlets appeared in the Netherlands, written mostly by greedy and ignorant quacks. The fact that the febrile attack was usually terminated by a critical sweat led them to the conclusion that an artificial production of profuse sweating would favorably terminate the disease. Hence it was recommended that the sick should be smothered in heavy featherbeds; that, all chinks and apertures of the bedroom being carefully stopped, a huge fire should be started in the chimney, and that heating drinks should be copiously administered. This procedure became known as the "Netherlands Regimen."

Soon, however, primarily through the efforts of a protestant English gentleman, Dr. Barr, who lived in Lübeck, and who was a learned man and a philanthropist, information about the English method of treatment filtered into public knowledge, and this led to the abandonment of the execrable and murderous "Dutch Regimen." Featherbeds were condemned and more rational, less active measures were adopted. Nevertheless, we see curious preoccupations persistently cropping up. One of them was that if the patient lapsed into sleep, he was irremediably lost. Evidently sleep and the deathly sopor of fatal coma were confounded. Hence heroic, but nevertheless ridiculous and useless efforts were made to prevent sleep. In the otherwise useful broadsheet of an unknown author printed at Wittenberg—the text, written in the vigorous and pithy vernacular of the sixteenth century, is well worth reading"—we are told: "Item.—Two men should have charge of the patient, to prevent his uncovering himself and his falling asleep. Item.—The men should watch and prevent sleep. Should, they not bewaring, the patient fall asleep, then he will lose his senses and will go mad. To ward off sleep apply rosewater to the tender parts of eyes and ears with a sponge or cloth, or better still put strong wine or sharp vinegar into his nose, talking to him all the while that he may stay awake. Item.—As to those who lie naked, should they refuse to keep still, then sew them up in the sheets with needle and thread and sew the sheets to the mattress, so that no air should get to them. Then spread the blanket."

Urtius Damianus, a popular practitioner of Ghent, went much further, in recommending that, the ordinary means for preventing sleep being ineffectual, single hairs should be plucked off the patient's head or beard, his members be forcibly and painfully constricted, and vinegar be instilled into the conjunctival sac. Most of the authors, however, advised against the use of sharp measures, such as vomitives, purges, venesection and the heating Dutch treatment, preferring what we would call nowadays the expectant method.

From this abundance of sources it is not difficult to construct a fairly precise clinical picture of the sweating sickness. Singly taken, the authors are not free from the ignorance of their age. One of them [11] saves himself all trouble by stating "The signs of the disease are manifest; those omitted might be reproduced from the reader's imagination." But taken altogether, the testimony of these continental authors represents a great advance over the paucity of the English.

[10] Hecker: pp. 129-130.

[11] Schiller, Joachimi ab Herderen, physici: De peste Brittannica, etc., Basileæ, 1531.

The unanimous assertion that the duration of the fever is precisely 24 hours need not be taken literally, for several of the writers admit variations. Thus we see Schiller saying "Habet inconstantes notas morbus," and Damian, "Diversus diversimode adoritur." I lay stress on this reflection for a reason that will became apparent later on.

The onset began with a chill, followed by very high fever, the patient frequently lapsing into coma. The chill was inaugurated with formication of the hands and feet, painful sensations under the fingernails, and deep prostration. Intense headache and backache, anorexia and vomiting, "broken-bone pains" of the limbs, later on delirium and coma, were the ordinary features. Dyspnœa, with cyanosis, set in with unconsciousness and generally led to death. The pulse was throughout very high and gradually became weaker; often heart-failure ended the process at the acme of the fever. After the lapse of 24 hours, the surface of the body became bathed in a most profuse and fetid perspiration. The action of the bowels and of the kidneys also showed variations. Sometimes it remained normal, then again it was suppressed. Exanthems: A vesicular eruption like our prickly heat (sudamina) is occasionally mentioned. Fatal cases, on the other hand, showed "bullæ, petechiæ, rhonchæ et pustulæ." The fear of suppressing perspiration was universal, a phantom that has survived until this day. Relapses were frequent, if not the universal rule, but each relapse was looked upon as a new invasion of the disease, the embodiment of which was conceived to reside in the single febrile attack. John Kaye mentions a case in which 12 were noted. Of autopsies there is no record. In England, as well as on the Continent, we meet with the assertion that the disease attacked mostly persons of middle age, and that children and old people were spared. Not one of the observers lays any stress upon the obvious fact that the ravages of the disease were most prominent among those enfeebled by want and privation, who naturally were the most subject also to excessive crowding in filthy dwellings.

The sweating sickness appeared in England for the fifth and last time at Shrewsbury on April 15, 1551. From here the malady spread again over the realm, diminishing gradually until the end of September, when it ceased. The morbidity was unexampled, the mortality appalling. Within a few days 960 of the inhabitants had died. City dwellers fled to the country, farmers from the country to the town. Many escaped to Scotland, Ireland, and to the Continent; daily labor ceased and the tolling of mortuary bells sounded uninterruptedly day and night. The loss of life was very great. No layer of society was spared. In this instance, again the fact is restated by John Kaye,[12] that the disease followed Englishmen wherever they went, not affecting, however, the natives. "And it so followed the Englishmen, that such merchants of England, as were in Flaunders and Spaine, and other countries beyond the sea were visited therewithall, and none other nation injected therewith." (Page 30.) This assertion is supplemented by another, according to which foreigners living in England remained immune. During this epidemic a curious phenomenon previously observed during the second invasion reappeared in England. Moulds of various colors, mainly red, but also white, yellow, ashy and black, became established on roofs, on clothing in use, even on stomachers and veils actually worn by women. Clothes kept in presses were spoiled, and utensils, articles of food fresh and preserved, were tainted by it. Frequently rain fell that was fringed with this mould, giving color to legends of rains of blood.[13]

This last invasion of the sweating sickness is notable for the fact, also, that it produced the first English medical publication dealing with it. The author was no less a man than John Kaye, the Caius of Gonville Hall, later Gonville College, now known as Caius College. At Henry VIII's request he lectured to the London Surgeons on anatomy. He became the physician of Edward VI, and was retained in this capacity by Queens Mary and Elizabeth. He became Linacre's successor in the presidential chair of the College of Physicians, and endowed two annual public autopsies, the first ones practised in England. He witnessed the epidemic of 1551, both at Shrewsbury and in London. His pamphlet dealing with it appeared in 1552. It is written in the forcible vernacular of the day, frankly condemning the coarse feeding and drunken habits of his countrymen; it eschews all learned verbiage, reserving erudition for the Latin edition of the opuscle. Like all his contemporaries, he attributed the malady to the effect of poisoned air. As a prophylactic, he recommends the use of cleansing bonfires, and supports the advice by the statement that cooks and blacksmiths showed a remarkable immunity. His treatment consisted of mild methods well established in England during the preceding epidemics. He deprecated all drastics, and condemned the forcing of perspiration, and the cruelties we read about in Damian. He was a versatile author, as attested by writings on subjects of natural history, which were dedicated to Conrad Gesner (among these is a volume "De canibus britannicis"), and by essays on historical and antiquarian matters. He died in 1573, ordering his epitaph to be: "Fui Cajus."

It is about 20 years ago that I came across and read Hecker's remarkable monograph on the sweating sickness. Intense interest was heightened by the puzzling uncertainty regarding the identity of this disease, which was dispelled only by acquaintance with the characteristics of the recurrent or relapsing fevers of modern times. By re-reading Hecker several times, and by employing the method of exclusion, I finally came to the conclusion that the sweating sickness observed in England and on the Continent in the fifteenth and sixteenth centuries must have been one of the virulent forms of relapsing fever, such as is rarely observed nowadays. The essential points are congruent, common factors being an infection carried by living parasites to human beings crowded together in filthy abodes, and as a predisposing

[12] Caius, John: A book or Counceill against the Disease commonly called the Sweate or Sweatyng Sickness, London, 1522.

[13] The chroniclers called this phenomenon "Lepra vestium."

element an enfeebled state of health caused by hunger, exposure and moral depression.

Thanks to Obermeyer we know that the causal parasite is the spirochæta febris recurrentis, which reaches the blood by various accidental forms of inoculation. The carriers of the disease are mainly head and body lice, ticks and possibly also the bedbug. In Europe it is to-day, as it must have been in England in the fifteenth and sixteenth centuries, a disease of the lousy. Nicholle has shown, in 1912, that lice hatched from eggs of infected lice are also infected. The same thing has been established regarding the eggs of the tick. Africa is the home of tick fever, while the pedicular forms are endemic in India, Central Asia and Eastern Europe. There is doubt as to the exact modes of inoculation. The entrance of the spirochæta was first assumed to be marked by the bite of the insect, but this is now questioned. As the spirochæta and its spores are mainly found in the intestinal tract of the insect, inoculation by the bite is unlikely. Experiments have demonstrated that the point was well taken; but if the debris of the crushed insect is rubbed into a fresh scratch, infection invariably follows. The capriciousness of the spread of the disease, formerly so unaccountable and puzzling, is readily explained by the capricious vicissitudes to which are subject the rise and fall of pedicular proliferation. The conditions parallel the puzzles presented by the transmission of malarial and yellow fevers, typhus, the plague, etc., by which older medical practitioners were sorely mystified.

In comparing the natural history of recurrent fever as observed in our day with the composite picture yielded by the writers who saw the sweating sickness of the fifteenth and sixteenth centuries, we find a general similarity of characteristics. But there are discrepancies which deserve attention. One is the statement that invariably 24 hours after the onset the fever was resolved by profuse critical sweating. Modern observers have found, on the other hand, that the duration of the febrile attack between initial chill and crisis is from 3 to 10 days. We have quoted two writers, Schiller and Damian, who explicitly mention the great diversity and inconstance of the features of the disease. Considerable variations in duration and virulence are noted even to-day in various epidemics, and but for the sheet anchor of the blood test some of the more atypical might even now not be easily identified. The fact that the writers have looked upon the individual febrile attack with its saliencies as the sum total of the disease, failing to recognize the essential importance of the tendency to recurrence, is not to be wondered at. The validity of the statements referring to the sudden cessation of the epidemics at the end of summer cannot well be questioned. All, with the exception of the first one, began in April or May and ended by October; therefore, they must be declared to have been summer weather epidemics—summer weather, to be sure, that was very damp and cool. It is interesting to compare this fact with the predilection of typhus fever, another disease of lousiness, for the winter months. The explanation may be found perhaps in the consideration that the inclemencies of the summer weather and the want prevailing during these epidemics had produced conditions almost identical with those that determine the hibernal eruptions of spotted fever. More difficult to explain are the assertions regarding the selective tendency of the sweating sickness, which is indicated in the statement that it followed Englishmen abroad, sparing the natives; foreigners living in England remaining also immune during the worst times of general sickness. The one great exception to this rule is found in the murderous sweep made by the malady through Northern Europe. Nevertheless, the repeated mention of this peculiarity must induce the reflection that the level of the personal habits of the generality of Englishmen of those days must have been below that of the continentals, a surmise of not much significance at a time of universal hygienic ignorance and neglect. As to the nature of the sweat exuded at the crisis and its excessive fetidity, contemporary statements are also unequivocal and unanimous. The sweat of rheumatic fever, having a peculiarly disagreeable acridity of odor, might be compared with it, but nobody would think of ascribing to this characteristic such stress as is done by these authors. The universal terror, the continuous spreading of rumors and the accounts of quick death, sweeping away masses of seemingly healthy people within a few hours, fear heightened by ignorance and superstition, must undoubtedly have created a general state of mind prone to overstatement and wild exaggeration. In times of stress and danger caused by war and pestilence nothing appeals more to the vulgar mind, ignorant or learned, than vehement assertion of the monstrous and ordinarily unthinkable; and nothing is more readily and passionately accepted. Each of the modern wars, the present one included, has furnished thousands of illustrations of this tendency. Once pronounced and vociferously repeated, such statements without adequate proof, become matters of universal belief and dogma.

SYNONYMS.

Febris sudatoria; morbus Anglicus; morbus sudatorius; pestis Brittannica.

The Sweat; Sweating Sickness.

La Suette.

De Schwetende Sicke (Platt).

Englicher Schweiss; Schweissucht; Schweiss-Krankheit; Hungerpest.

Schwitzende Seuche.

Der Engelske Sved.

Engelske Svetten.

Ἱδρωνοῦσοι; ἱδρωπύρετοι.

JOHNS HOPKINS HOSPITAL BULLETIN.

The Hospital Bulletin contains details of hospital and dispensary practice, abstracts of papers read and other proceedings of the Medical Society of the Hospital, reports of lectures, and other matters of general interest in connection with the work of the Hospital. It is issued monthly. Volume XXVII is now in progress. The subscription price is $2.00 per year. (Foreign postage, 50 cents.) Price of cloth-bound volumes, $2.50 each.

A complete index to Vols. I-XVI of the Bulletin has been issued. Price 50 cents, bound in cloth.

NOTES ON NEW BOOKS.

Pellagra: An American Problem. By GEORGE M. NILES, M. D., Atlanta, Ga. Second edition. Illustrated. $3.00. (*Philadelphia and London: W. B. Saunders Company, 1916.*)

The appearance of the second edition of this book is timely, in view of the large additions to our knowledge of pellagra during the past four years. The author approves Lombroso's dictum "In pellagra we are dealing with an intoxication produced by poisons developed in spoiled corn through the action of certain micro-organisms in themselves harmless to men," but would add to the words "spoiled corn" "spoiled carbohydrates."

The chapter entitled "General Considerations, Historic and Otherwise" presents an excellent résumé of the history of the disease since its first discovery. In the account of "Pellagra in the United States" we notice that he fails to mention a case reported by Babcock in the *American Journal of Insanity*, July, 1912, with the facsimile of the notes on a patient admitted to the Columbia State Hospital, February 14, 1834. This seems to have been an undoubted case. The other chapters of most interest relate to the etiology and treatment of the disease. The chapter giving details of experimental work is inconclusive and unsatisfactory. The book as a whole presents an excellent account of our present knowledge of pellagra.

Nervous Children: Prevention and Management. By BEVERLEY R. TUCKER, M. D., Professor of Neurology and Psychiatry, Medical College of Virginia. $1.25. (*Boston: Richard G. Badger, 1916.*)

This book of 147 pages is "to be read with equal interest," according to the hope of the author, "by the physician, the school teacher, the mother and the intelligent nurse." It is clearly written and attractively printed. It presents little that is new, but portions of it seem well adapted to the instruction of well-intentioned parents.

Diseases of the Arteries Including Angina Pectoris. By SIR CLIFFORD ALLBUTT, K. C. B., M. A., M. D., F. R. C. P., F. R. S., Hon. M. D., LL. D., D. Sc., etc., Regius Professor of Physic in the University of Cambridge, Fellow of Gonville and Caius College, Hon. Fellow New York Acad. Med., etc. In two volumes. $9.00. (*London: Macmillan Company, Limited, St. Martin's Street, 1915.*)

To him who has once ventured into the dark in its pursuit, arterio-sclerosis is the will-o'-the-wisp of medicine, appearing now here and now there with seeming lawlessness, its explanation in one case grasped, yet in the next seeming more far away than ever. Elusive, tantalizing, its pursuit exercises an irresistible fascination over all who have allowed themselves to be drawn into it. These are a goodly company, with many of the best minds that have studied medical problems from the viewpoint of pathologist or physiologist or clinician. Among them Sir Clifford Allbutt, through a long life, has again and again turned to this quest, now in the laboratory, now at the bedside, and through it all, in his library, has been amassing a tremendous knowledge of the phenomena of arterial disease seen by other eyes than his own, and of the views, outworn and current, advanced by other workers in explanation of their causation. Now, rich beyond the usual lot of men in years, in knowledge and in experience, he has brought together in two volumes the results of the favorite labors of his life.

These volumes contain such a mass of facts and so painstaking and compendious a recital of argument on all phases of these oft-debated subjects that it is impossible to review them in detail. Beginning with an historical introduction, the subject is approached first from the standpoint of the physics of the circulation, in a very complete chapter, followed by an interesting discussion of the viscosity of the blood. Then arterio-sclerosis is considered from the standpoint of its many possible and probable causes in a chapter of 154 pages. The author closes with the following paragraph:

"In revising this chapter on the Causes of Arterio-sclerosis I sympathise with a reviewer who, after reading a certain dissertation on another disease, exclaimed, 'We would ask what the conditions are which do not bring on this malady!' And every failing element is both cause and effect."

Nevertheless, he does lead the reader to a viewpoint from which the otherwise hopeless tangle of fact and theory which he presents may be seen to take on definite form and order. This viewpoint envisages arterio-sclerosis as an omnibus name crudely signifying the anatomical result of several morbid processes, not as a disease, a definite etiological entity. For him arterio-sclerosis is of two main types. One associated with, and caused by, high arterial pressure he names "hyperpietic"; the other, a degenerative lesion seen typically in the aged, dependent upon various infectious, toxic and other unknown factors, he distinguishes as "decrescent" arterio-sclerosis. This view, in the main, he has long preached. The term "decrescent" he has substituted for the word "involutionary," formerly used by him in the same sense, but which he considers less exactly descriptive. This viewpoint is coming to be very generally held by students of the subject, and the reviewer is in entire sympathy with it. Faber, in his recent monograph, endeavors to bring a further unity of the two types in a mechanical conception of disproportion between internal strain and strength of the vessel-wall. It is to be hoped that physicians generally may acquire some such point of view and give the diagnosis of arterio-sclerosis, as the designation of a disease embracing everything from syphilis of the aorta to chronic Bright's disease, a much-needed rest. The reviewer doubts, however, whether the rather pedantic terms "hyperpietic" and "decrescent" will do much toward popularizing the underlying ideas.

There follow in the first volume quite elaborate presentations of the relation of arterio-sclerosis to disease of the kidneys, of the symptoms and the morbid anatomy of arterio-sclerosis. The second volume continues with cardio-sclerosis and the effects of arterio-sclerosis and hypertension upon the heart, then a chapter upon diagnosis and prognosis which is curiously brief when contrasted with what has gone before, and is not particularly authoritative. The author feels unable to formulate any principles for the very important practical application of prognosis in life insurance. The chapter on treatment is full and contains much sane and valuable advice.

In Part Two he deals with aortitis and angina pectoris in the same complete and painstaking way. Chapter Six is a very valuable summary of all of the hypotheses that have been advanced for the explanation of the anginal paroxysm, covering 140 pages. His own belief, held for many years, that angina pectoris is fundamentally dependent upon disease of the wall of the ascending aorta, is only confirmed by his final review of the evidence. On page 422 of Volume Two he says:

"I have failed to discover a case of unmistakable angina in which, whatever the disease of the coronary arteries, the aorta, on careful examination, was demonstrated histologically to be inwardly and outwardly sound."

Whatever the final explanation of the whole mechanism of angina pectoris, Allbutt's theory has certainly received considerable confirmation from recent studies of syphilis of the aorta.

The concluding chapters on diagnosis, prognosis and treatment are good. The reviewer would call attention to a small detail to emphasize a fact not widely known, and evidently not appreciated

by the author. He states on page 534 that nitroglycerin by the mouth acts in about 10 minutes. This is true if it be swallowed. Do not, therefore, conclude that nitroglycerin acts too slowly to relieve an anginal paroxysm. Nitroglycerin should never be swallowed, but should be held in the mouth until absorbed, which requires somewhere between 45 seconds and a minute and a half, at the end of which time its systemic effects can almost invariably be detected.

A reviewer bent on hostile criticism might hold that the book should have been named "An Autobiographic Sketch of the Diseases of the Arteries." Those of us who know Sir Clifford Allbutt and his work; who appreciate his early recognition of syphilis of the vascular system, well in advance of its more complete description by the Kiel pathologist, and far in advance of contemporary medical opinion; who recognize the great weight of his authority in popularizing the use of instruments for measuring blood pressure, and of more exact physiological concepts in circulatory disease; who find his views on arterio-sclerosis and angina pectoris receiving more confirmation with the years, cannot but value very highly his own account of his researches and of the development of these views in his own mind. As a reference-book for the ready location of a fact or theory, the book has the serious defects of its author's well-known style. Those of us who do not agree with Sir Clifford that the ideals of scientific presentation and of artistic portrayal in literature are identical, cannot but regret that the storehouse of his erudition was not made more readily accessible to English-speaking workers in this field of which he is a master; but all of us who regard him with admiration and affection must admit his right to open that storehouse to us on his own terms and must express our debt of gratitude for his generosity with its contents.

THEODORE C. JANEWAY.

Local and Regional Anæsthesia. By CARROLL W. ALLEN, M. D. $6.00. (*Philadelphia: W. B. Saunders Company, 1914.*)

This volume of 600 pages is the result of 20 years of earnest and laborious work on local and regional anæsthesia. The introduction is written by Prof. Rudolph Matas, to whom the volume is dedicated.

In the opening chapter the author gives a brief but interesting history of the development of local anæsthesia. Succeeding chapters deal with the sensations, especially pain, and contain a full discussion of the theories of pain. Under "Osmosis and Diffusion" the author points out the importance of using solutions of proper tonicity, in order that a minimal amount of pain may be produced and the best conditions for healing may be obtained. He gives a complete list of all the local anæsthetics known, and discusses fully their chemistry, toxicity and effect upon tissues (local), the duration of anæsthesia, etc. He concludes that novocaine is "at the present time the most commendable of local anæsthetics, and combined with adrenaline its value is greatly enhanced." The principles underlying the technic of administration are dealt with in proper detail.

The greater portion of the book is devoted to topographical anæsthesia. There are numerous diagrams, charts, and photographs representing anatomical dissections of various parts of the body, for the purpose of demonstrating the proper points and lines for cutaneous and deep infiltration.

Great care has been exercised in determining these various areas, and no portion of the body has been neglected.

The book is an exhaustive treatise on this subject of local and regional anæsthesia, but owing to its great volume and detail will be of value chiefly as a reference book, and to this end it should serve a great need. The hope of its author, however, will not be fulfilled, unless it is instrumental in diminishing the indications for general anæsthesia.

C. H. W.

The Alimentary Tract: A Radiographic Study. By ALFRED E. BARCLAY, M. A., M. D. Cloth, $4.00. (*New York: Macmillan Company, 1915.*)

The first edition of Barclay's book on the radioscopic study of the gastro-intestinal tract was received with so much favor that a second edition became necessary within a year's time. In the present volume the author, besides making many additions to the subject matter considered in the first printing, has added some new chapters of very pertinent interest, notably one upon the etiology of gastric and duodenal ulcer and another upon various questions involved in the physiology and pathology of the large bowel. The conclusions are based entirely upon fluoroscopic studies, and this is of fundamental importance, because it calls attention to the fact that in the study of gastro-intestinal pathology, where as a rule function is so much more important than form, far better conclusions as to the underlying conditions may be obtained from the more plastic radioscopy than from the less plastic radiography, unless in the latter instance the case be studied cinematographically. That the study of the plates is so much more widely employed than the use of the screen in the digestive field is unfortunate, but it has been brought about by many factors, chief among which are the fear of the screen on the part of many operators and the far greater difficulties of technique. On the other hand, given an absolutely satisfactory apparatus and proper training in interpretation of the screen shadow, that the results obtained by fluoroscopy are more satisfactory seems to us to be beyond peradventure. The fact that we can study the patient in upright and in prone positions and that we can note the effect of respiratory movements and massage seem to us sufficient reasons for making this the method of election; and Barclay's extremely valuable contributions to gastro-intestinal pathology, based entirely upon fluoroscopic studies, accentuates this point of view.

The early pages of the book are devoted to discussions of technique, protection and the most satisfactory routine methods to be employed. Next, we find chapters on the œsophagus, the normal and pathological stomach, gastric ulcer, hour-glass stomach, gastric carcinoma and various other pathological gastric conditions, including a very interesting chapter upon the etiology of gastric and duodenal ulcer. Following these are chapters upon the small and large intestine in health and disease, the latter part of the book being devoted to an epitome of the cases considered and a very complete bibliography. Not the least interesting features of the book are the author's philosophical remarks, based upon these fluoroscopic studies, regarding various tendencies of the day in gastro-intestinal pathology and therapy. For example, "'What is one man's food is another man's poison' is an old saying that expresses the fact that this complex nervous mechanism controlling the digestive tract is not standardized; that each individual is a separate creation, each bearing outward resemblance of a standard article. Even the gross appearances of a normal stomach vary in such a way that it is only a study of a large number that will give a true conception of the variations that are possible within the limits of the normal." Again, "If we could accept the universal 24-hour standard, the difficulties of writing on this subject would be comparatively slight, and dogmatism could be indulged; but since each case must be treated as an individual, the evidence deduced from watching the passage of a standard meal must be most carefully balanced with the clinical picture, before deductions are made." And finally, apropos of the much discussed colonic resection for intestinal auto-intoxication, "The fact that certain wholesale removals are successful (and this undoubtedly is true in a small percentage of cases) is no proof that they were necessary, but rather that somewhere in this large mass lay the cause of trouble"; and, "One is astonished, not so much at the brilliancy of the surgery displayed, as at the tolerance of the human body."

In each problem presented Barclay makes his attack from the physiological or functional, as well as from the anatomical or morphological, point of view; and this adds immensely to the value of his subsequent conclusions. We feel that this book affords the best presentation of the brief in favor of fluoroscopy in the diagnosis of gastro-intestinal diseases, and we can recommend it heartily not only to the special students in this field, but to every one interested in digestive pathology. F. H. B.

X-Rays: How to Produce and Interpret Them. By HAROLD MOWAT, M. D. (Edin.). Cloth, $3.00. (*London: Oxford University Press, 1915.*)

The author has described in a clear and concise way the principles pertaining to the physics of x-rays, and especially the different sources of current capable of exciting the x-ray tube.

The chapter on the technique of examination and development of x-ray negatives is well worthy of mention, and should be very helpful to the beginner in roentgenology.

The remaining half of the book is devoted to interpretations of the plates and findings with the fluoroscopic method of examination. It takes up the question of the different changes seen on the plate and describes those that are most typical; but the text is far too brief and good roentgenograms for comparison are lacking.

On the whole, the book would only be suitable for a beginner or to one who does not expect to devote his entire time to the study of roentgenology. C. A. W.

A Text-Book upon the Pathogenic Bacteria and Protozoa. By JOSEPH MCFARLAND, M. D., Sc. D. $4.00. (*Philadelphia: W. B. Saunders Company, 1916.*)

The fact that McFarland's text-book on bacteriology has passed through seven editions since its first appearance in 1896 is indisputable evidence of the esteem in which it is held by physicians and medical students. The present eighth edition comes to us in a volume of some 800 pages embodying the subject matter of bacteriology and protozoology in its widest sense. Not much need be said here that has not already been said of previous editions, since, while considerably altered in arrangement and somewhat changed in text, the book does not present any very noticeable alteration in its general treatment of the subject. Just as in earlier editions, the place of Koch in bacteriology is incorrectly represented. A reading of the historical chapter gives one the impression that the contributions of Koch were more or less logical amplifications and extensions of earlier investigation. We know, both from the effect of Koch's work on the development of the science of bacteriology, as clearly seen in the literature of the subject, and from the evidence of Koch's own contemporaries, many of whom are still living and active, that his work was revolutionary, and that the methods devised by him raised the science of bacteriology to a new plane from which its modern development began. The book is also full of mistakes. Thus, on page 458, one finds the statement that the pneumobacillus of Friedländer "may belong to the same group in which we find *Bacillus aërogenes capsulatus.*" Manifestly, this statement can only be correct if we substitute the name *B. lactis aërogenes* for *B. aërogenes capsulatus.* On page 589 we are informed that the typhoid bacillus "finds abundant opportunity for growth and development, etc., and can be found in water, soiled clothing, dust, sewage, milk, etc., contaminated directly or indirectly with the intestinal discharge of diseased persons." References to investigations which substantiate this assertion are lacking, and without such references the statement may properly be challenged. On page 616 the statement is made that *B. coli* "may also be identical with *B. lactis aërogenes,*" about which one may express reasonable doubt; and on page 625 the hog-cholera bacillus (*B. suipestifer* of Salmon and Smith) is described as fermenting lactose, which it does not do. Finally, on page 732, in the chapter on Actinomycosis, no adequate reference is made to Wright's work, altogether the most important done for some years in this field, although four of the seven illustrations are taken directly from Wright's paper.

McFarland's text-book, as we see it, contains a vast amount of valuable information. It is still in need of thorough revision, however, in the course of which the author should apply the pruning knife liberally, cut out the many misstatements and generally bring the book up to the standard set by our other text-books of bacteriology and protozoology in this country.

Circulation in Health and Disease. By CARL J. WIGGERS, M. D. (*Philadelphia: Lea & Febiger, 1915.*)

It is proper that at definite intervals in the large book that records the development of a scientific subject there should be a summarizing chapter, in which a critical opinion should be expressed concerning the preceding work. Cardiac physiology, and as a result cardiac pathology, have made most wonderful advances in the past decennium. This is well appreciated in considering our present knowledge of the arrhythmias and comparing it with that of 10 years ago, when the electro-cardiograph first came into general use. But such rapid strides are dangerous; they must be controlled carefully and dispassionately.

In his recent book on the Modern Aspects of the Circulation in Health and Disease, Dr. Carl J. Wiggers, in 350 pages, reviews an abundant literature well chosen and collected at each chapter's end, and colors his conclusions with the rich experience obtained in the physiological laboratory. The treatise is divided into three sections: Physiology of the Circulation, Graphic Methods for the Clinician, and Diseases of the Circulation. The last section briefly outlines the physiology of the diseases of the circulation, following the method of Krehl who is frequently quoted. The first two sections are especially to be commended, and will be read with interest by all cardiac investigators. The book is technical and perhaps the conclusions are somewhat dogmatic, but the suggestions and outlines for new work are abundant, and show clearly the careful consideration of a thoroughly trained laboratory worker. The criticism of the mechanics of the different "instruments of precision" is most instructive, and emphasizes the dangers of regarding laboratory information as necessarily conclusive. The book will hardly be suitable for daily use by the general practitioner, but is essential for the library of the "whole time" student and teacher. E. W. B.

The Book of the Fly: a Nature Study of the House-Fly and Its Kin; the Fly Plague and a Cure. By G. HURLSTONE HARDY. 80 cents. (*New York: Rebman Company.*)

This book gives in a popular way a few points on the identification of life histories of house-flies and some of the commoner species of outdoor flies. There is a short chapter on a family of flies which may go through part of their life cycle within the bodies of animals and occasionally of man. The subject of the dissemination of disease by flies is briefly touched upon, and new methods of conducting an effective warfare against them are suggested. An appendix gives a minute description of the structure of the fly and a list of 60 families with their distinguishing characteristics. The book is written for the general public rather than for the medical profession. M. C.

The Gold-headed Cane. By WILLIAM MACMICHAEL, M. D. With an Introduction by SIR WILLIAM OSLER, B. A., M. D., F. R. S., and a Preface by FRANCIS R. PACKARD, M. D. (*New York: Paul Hoeber, 1915.*)

American physicians are to be congratulated on the opportunity to secure at a small price this admirable reprint of one of the Classics of Medical History.

Perhaps no other single volume presents so attractive a picture of the clinical traditions of the great English physicians as this little volume published first anonymously by MacMichael in 1827, and in an enlarged second edition in 1828.

It is this second edition which has been so beautifully reproduced with all the original cuts. Its value is further enhanced by an introduction by Sir William Osler and a preface by Francis Packard, who gives us an excellent sketch of the life of the author. The publisher, Hoeber of New York, deserves the thanks and encouragement of the profession.

Nurses' Handbook of Drugs and Solutions. By JULIA C. STIMSON, R. N. $1.00. (*Boston: Whitcomb & Barrows, 1915.*)

This Nurses' Handbook of Drugs and Solutions is essentially what the title implies. The preface intimates that it is also intended as a text-book in training-schools for nurses; but for this purpose it is too brief and would be wholly inadequate, on account of the lack of detailed information which it is essential for a nurse to know in order that she may work intelligently.

As a handbook, however, it should serve a very useful purpose. It is conveniently small, clearly printed, and deals with all of the more important drugs. The chapter on Weights, Drugs and Solutions is clearly and briefly written, and should prove valuable for reference. S.

A Guide to Gynecology in General Practice. By COMYNS BERKELEY, M. A., M. D., etc., and VICTOR BONNEY, M. S., M. D., etc. Cloth, $6.50. (*New York: Oxford University Press, American Branch.*)

The authors of this work are already known to the American profession through the publication in this country five years ago of their Text-Book of Gynecological Surgery, which was devoted wholly to the operative side of gynecology. The present treatise omits all details of operative technique. It is confined almost entirely to the medical side of this specialty, embracing in elaborate detail both general and special gynecological diagnosis, as well as non-surgical therapeutic measures. In other words, in America, it would be called a *medical gynecology.*

The subject matter is presented in five parts. The first of these describes the methods employed in making a gynecological examination. The second undertakes to instruct the student in the proper significance of gynecological symptoms. The third is addressed in a similar way to the interpretation of the physical signs encountered in the various pelvic disorders in women. The fourth has to do with treatment, and is accorded one-fourth of the entire text, detailed advice being given in all conditions amenable to non-surgical therapy. Finally, in part five, the medico-legal aspect of gynecology is presented in a most practical and readable form.

The index is unusually complete and is made especially attractive through the liberal use of bold-faced type for the sub-heads. This splendid index, together with an elaborate system of cross-references profusely scattered throughout the text, render immediately accessible any desired subject. The illustrations are of mediocre quality, but the publisher's work has been well done.

The book may be safely recommended, both to student and practitioner, as being a safe and helpful guide in this field.

E. H. R.

Practical Cystoscopy and the Diagnosis of Surgical Diseases of the Kidneys and Urinary Bladder. By PAUL M. PILCHER, A. M., M. D. Second edition. Cloth, $6.00. (*Philadelphia and London: W. B. Saunders Company, 1915.*)

During the four years that have elapsed since the first appearance of this book urological studies have been prosecuted with extraordinary intensiveness. How fruitful this labor has been, is made strikingly evident by a comparison of the two editions.

The original logical arrangement of subject-matter has been retained, but with important revisions, amplifications and additions. Thus, an entire new section has been devoted to pyelography, appended to which is a series of illustrative radiograms contributed by Dr. Wm. F. Braasch, of the Mayo clinic. It is unfortunate that the value of thorium nitrate, recently discovered by Dr. J. E. Burns of The Johns Hopkins Hospital urological staff, and recommended as an ideal substance for pyelography, was not sufficiently well established at the time of the publication to be included in this section. It will undoubtedly replace all other substances used for this purpose. The chapters on disease of the prostate and on functional activity of the kidneys have been entirely rewritten so as to incorporate the more recent advances. Notable additions are to be found, too, in the section dealing with the therapeutic use of the cystoscope, special attention having been properly given to the use of the high frequency current in bladder conditions.

The book can be recommended, alike to specialist and practitioner, as a splendid presentation of the entire subject of cystoscopy. E. H. R.

An Introduction to Bacteriology for Nurses. By HARRY W. CAREY, A. B., M. D. (*Philadelphia: F. A. Davis Company, Publishers, 1915.*)

In this little book of 133 pages the author succeeds admirably in his avowed purpose of presenting clearly and in simple language the portion of the subject essential for the nurse to know.

It is based upon the lecture notes of the author after an experience of eight years in the teaching of nurses and may be highly recommended. T. P. L.

Materia Medica for Nurses. By A. S. BLUMGARTEN, M. D. (*New York: The Macmillan Company, 1914.*)

Blumgarten's Materia Medica does not seem especially suitable as a text-book in training-schools for nurses. The information contained in the book could have been very much condensed, thus avoiding repetition and making the book a more convenient size.

The language used is clear and simple and the definitions are well-worded. The classification of drugs is also good; but there is a good deal of repetition.

Information concerning the anatomy and physiology of the various organs of the body could hardly be considered as of great value in a text-book on materia medica.

The chapter on solutions is somewhat complicated and its usefulness would seem to depend entirely on the memorizing of arbitrary rules. As a reference book the work might be used to advantage. M. S.

Collected Papers of the Mayo Clinic, Rochester, Minn. Vol. VI. 1914. Edited by M. H. MELLISH. $5.50 net. (*Philadelphia: W. B. Saunders Company, 1915.*)

The papers are divided into six general parts: (a) Alimentary Canal (26), (b) Urinogenital Organs (13), (c) Ductless Glands (15), (d) Head, Trunk and Extremities (11), (e) Technic (8), (f) General Papers (5)—in all 78 papers by 28 authors.

In the first paper a plastic operation for repair of defect after removal of epithelioma of the lower lip is described for the first time. Many good x-ray pictures illustrate articles on gastric and duodenal lesions and a good description of the technic is given. Stress is laid on the relation of gastric ulcer to gastric cancer and the importance of early operation. A few illustrations give an idea of the types of operation used. Dr. Will Mayo reports three per cent of recurrences of symptoms after operations for duodenal ulcer—gastrojejunostomy having been the method of choice. He is convinced that a too coarse silk has been used in many anas-

tomoses, and he recommends a No. 0 silk. He believes that it is the silk in the outer layers of the suture that causes trouble by working into the lumen and, dangling partially free, prevents healing. He hesitates to use catgut in the outer layer as it is such a good culture medium.

Braasch reports that in 1000 patients in which uretero-pyelography has been employed there have been no fatalities and no permanent injury, 10 per cent collargol and the gravity method of injection of the warm solution being used.

Judd reports 218 cases of chronic mastitis and reviews the end results after operations for cancer of the breast. He believes that chronic cystic mastitis has a definite relation to cancer and in many instances may be considered a pre-cancerous lesion. In 7 out of 218 cases a complete breast operation with gland and muscle resection was done. He states that at times after the complete breast operation permanent limitations of movement and swelling in the arm and shoulder occur. May this not be due to lack of care in closing the wound, so that there is tension of skin over the new axilla, thus leaving a dead space around the vessels? This space fills with blood which in the presence of a low grade infection from the skin organizes and constricts the blood vessels, resulting in considerable scar and subsequent edema and lack of function, a point strongly emphasized by Dr. Halsted.

Beckman (in writing of decortication of the lung) reports a case of an old empyema, treated by decortication, in which the entire lung expanded after being totally collapsed for two years. Decortication produces less shock than the Schede operation.

One of the most interesting of the papers is that in which Wm. Mayo writes of clinics in Germany, Belgium and Great Britain which he visited in 1913.

BOOKS RECEIVED.

Cerebellar Abscess. Its Etiology, Pathology, Diagnosis and Treatment Including Anatomy and Physiology of the Cerebellum. By Isidore Friesner, M. D., and Alfred Braun, M. D., F. A. C. S. With 10 full page plates and 16 illustrations in text. 1916. 8°. 186 pages. Paul B. Hoeber, New York.

The Diagnosis and Treatment of Heart Disease. Practical Points for Students and Practitioners. By E. M. Brockbank, M. D. (Vict.), F. R. C. P. Second edition, with illustrations. 1916. 12°. 120 pages. Paul B. Hoeber, New York.

Clinical Disorders of the Heart Beat. A Handbook for Practitioners and Students. By Thomas Lewis, M. D., D. Sc., F. R. C. P. Third edition. 1916. 8°. 116 pages. Paul B. Hoeber, New York.

Modern Medicine and Some Modern Remedies. Practical Notes for the General Practitioner. By Thomas Bodley Scott. With a Preface by Sir Lauder Brunton, Bart., F. R. S. 1916. 12°. 159 pages. Paul B. Hoeber, New York.

Mentally Deficient Children; Their Treatment and Training. By G. E. Shuttleworth, B. A., M. D., and W. A. Potts, M. A., M. D. Fourth edition. 1916. 12°. 284 pages. P. Blakiston's Son & Co., Philadelphia.

The Primary Lung Focus of Tuberculosis in Children. By Dr. Anthon Ghon. English edition. Authorized translation by D. Barty King, M. A., M. D. (Edin.), M. R. C. P. (Lond. and Edin.). With seventy-two text illustrations, one black and one colored plate. 1916. 8°. 72 pages. Paul B. Hoeber, New York.

Localization by X-Rays and Stereoscopy. By Sir James Mackenzie Davidson, M. B., C. M. Aberd. With 35 stereoscopic illustrations on special plates and other figures in the text. 1916. 8°. 72 pages. Paul B. Hoeber, New York.

The Pathology of Tumors. By E. H. Kettle, M. D., B. S., Lond. With one hundred and twenty-six illustrations 1916. 8°. 224 pages. Paul B. Hoeber, New York.

New York Dermatological Society, 1869-1916. Constitution and By-laws. List of Members. 1916. 12°. 15 pages. New York.

Practical Massage and Corrective Exercises. By Hartvig Nissen. Revised and enlarged edition of the Author's "Practical Massage in Twenty Lessons," with many additions. With 68 original illustrations, including several full-page, half-tone plates. 1916. 12°. 211 pages. F. A. Davis Company, Philadelphia; Stanley Phillips, London.

Skin Cancer. By Henry H. Hazen, A. B., M. D. With ninety-seven text illustrations and one colored frontispiece. 1916. 8°. 251 pages. C. V. Mosby Company, St. Louis.

Diseases of the Digestive Tract and Their Treatment. By A. Everett Austin, A. M., M. D. With eighty-five illustrations, including ten color plates. 1916. 8°. 552 pages. C. V. Mosby Company, St. Louis.

Obstetrics, Normal and Operative. By George Peaslee Shears, B. S., M. D. 419 illustrations. 1916. 8°. 745 pages. J. B. Lippincott Company, Philadelphia and London.

Diseases of the Skin. By Richard L. Sutton, M. D. With six hundred and ninety-three illustrations and eight colored plates. 1916. 8°. 916 pages. C. V. Mosby Company, St. Louis.

The Dream Problem. By Dr. A. E. Maeder. Authorized translation by Drs. Frank Mead Hallock and Smith Ely Jelliffe. Nervous and Mental Disease Monograph Series No. 22. 1916. 8°. 43 pages. The Nervous and Mental Disease Publishing Company, New York.

Ohio State Board of Health. Twenty-ninth Annual Report of the State Board of Health of the State of Ohio for the Year Ending December 31, 1914. 1915. 8°. 894 pages. The F. J. Heer Printing Company, Columbus, Ohio.

United States Department of Commerce, Bureau of the Census. United States Life Tables 1910. Prepared under the supervision of Prof. W. Glover of the University of Michigan. 1916. 4°. 65 pages. Government Printing Office, Washington.

A Text-Book of Pathology. By W. G. MacCallum. With 575 illustrations, chiefly from the drawings of Alfred Feinberg. 1916. 8°. 1085 pages. W. B. Saunders Company, Philadelphia and London.

A Text-Book of Practical Gynecology. For Practitioners and Students. By D. Tod Gilliam, M. D., and Earl M. Gilliam, M. D. 5 rev. ed. Illustrated with 352 engravings, a colored frontispiece, and 13 full-page half-tone plates. 1916. 8°. 681 pages. F. A. Davis Company, Philadelphia; Stanley Phillips, London.

Harvey's Views on the Use of the Circulation of the Blood. By John G. Curtis, M. D., LL. D. Based on a Lecture Delivered in 1907 before The Johns Hopkins Hospital Historical Club at Baltimore. 1915. 12°. 194 pages. Columbia University Press, New York.

Christianity and Sex Problems. By Hugh Northcote, M. A. 2 ed., revised and enlarged. 1916. 8°. 478 pages. F. A. Davis Company, Philadelphia: Stanley Phillips, London.

The Johns Hopkins Hospital Bulletins are issued monthly. They are printed by the LORD BALTIMORE PRESS, Baltimore. Subscriptions, $2.00 a year (foreign postage, 50 cents), may be addressed to the publishers, THE JOHNS HOPKINS PRESS, BALTIMORE; single copies will be sent by mail for twenty-five cents each. Single copies may also be procured from the BALTIMORE NEWS CO., Baltimore.